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PREFACE.

In preparing this Compend of Special Pathology I have made use of Prof. James Ewing's Clinical Pathology of the Blood for the pages which deal with that subject, and the chapter on the Pathology of the Skin has been entirely written by Dr. J. C. Johnston, of Cornell Medical College. It gives me pleasure to thank these gentlemen for their valuable aid.

A. E. T.

CORNELL UNIVERSITY
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COMPEND OF PATHOLOGY.

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PART II. SPECIAL PATHOLOGY.

CHAPTER I.

CIRCULATORY ORGANS. I. THE HEART.

Malformations.

Cause.—Two sources of anomalous formation are known, either inhibition of normal development or disease during fetal life.

The heart is at first a straight tube with the primitive aorta at its anterior (or superior) end and the omphalo-mesenteric veins at the posterior (or inferior). Its rate of growth exceeds that of the cavity in which it lies, and hence it early becomes twisted, so that the arterial end lies superficial to the venous; further twisting and development lead to the formation of the auricles from the deeper limb of the tube, the beginning of the great arteries from the superficial limb, and the ventricles from the middle of the bow joining the two limbs. The tube is made up of an inner endothelial portion and an outer muscular. Septa between auricles and ventricles begin to appear (fourth week) as folds which meet and become continuous, earlier in the auricles; in the primitive aorta a spirally twisted septum divides the vessel into two more or less triangular blood channels; by the seventh week this division of heart and vessels is about complete.

Commonest among anomalies by defect are imperfect septa and malformed vessels. The entire heart may be lacking or rudimentary, or divided into only two chambers (fish type), or there may be one ventricle and two imperfect auricles (reptilian type), or either septum between the two sides of the heart may be imperfect with an undivided truncus arteriosus, or there may be atresia or stenosis of either aorta or pulmonary artery, the latter more commonly affected. Stenosis of the pulmonary, like persistence of the interauricular opening, causes extreme cyanosis, although not incompatible with life, but in the former case the morbus ceruleus is due to general venous stasis, while in the latter it is caused by mixture of venous with arterial blood and may not be very marked. Narrow slit-like communications between the auricles are far commoner than generally supposed and may give no symptoms (25–30 per cent. of all bodies examined). Lastly, the heart, or great vessels, or both may be transposed, and the abdominal organs may or may not show similar misplacement (situs inversus partialis and totalis). The ductus arteriosus (d. Botalli) may close too soon, from stenosis of the pulmonary, or may persist and is then usually associated with septum defects ; the persistence, if the only anomaly present, may depend upon imperfect filling of the lungs, irregular course or unusual diameter of the vessel. The cusps of the pulmonary and aortic valves may be increased or diminished by one. Imperfect development of the anterior thoracic wall or the diaphragm may lead to ectopia cordis though the heart itself is normally developed.

Disordered Circulation in Heart and Great Vessels.

Cardiac Thrombosis.—Cause as for thrombosis elsewhere, slow blood stream, loss of endothelium and roughened surfaces; the former especially where there are natural or pathological sinuses or trabeculæ which direct the stream, as either appendix auriculæ, between papillary muscles, in aneurysms, the latter especially with inflammation (endocarditis). Thrombi are found more often in the right side, in the auricles and apices of the ventricles; if between papillary muscles they show root-like processes; if recent they may be loosely attached as a yellowish-white film, if older they become firmly fixed (organized), the freshest still contain red cells and blood pigment. They form by the collection of leucocytes where the blood stream is slow, then blood plates collect, fibrin is deposited and the color is white, or streaked with red cells and their pigment; these elements, especially the white cells, degenerate and the thrombus may be washed away, or a cavity may form within the thrombus filled with puriform fluid. In the last hours of cachectic patients such thrombi form rapidly and reach a large size; they may be polypoid and fragments may break off and form emboli. In endocarditis small firm white thrombi are often found as vegetations on valves, especially in verrucous endocarditis.

The results of cardiac thrombosis are mechanical interference with the heart's action, narrowing of lumina and impediment to the circulation, embolism of distant arteries, abscess and perforation of the heart wall when the thrombus contains bacteria; softening or organization or calcification of the thrombus itself, the latter called *cardiolith*.

Disorders of the Coronary Arteries.—Protected by the cusps of the aortic valve, embolism seldom occurs. Thrombosis is common late in life and may be the cause of sudden death; such thrombi form usually where the lumen is already narrowed by sclerotic and atheromatous changes, and the symptoms of this condition, cardiac palpitation, arhythmic and intermittent pulse, angina pectoris, often precede the fatal termination over a period of months. If the thrombus forms very slowly a collateral circulation may prevent serious symptoms till some muscular or emotional crisis ends in sudden death. Although anastomoses occur between certain branches of the coronary arteries, these are so undeveloped that the vessels may be considered as end arteries through most of their distribution. At their origin from the aorta the coronary arteries are often narrowed by atheromatous plates in the aorta and such stenosis may be found even in young persons; up to a certain degree the heart is tolerant of this condition, but as its nutrition suffers in proportion to the stenosis, sudden cessation of function may occur at any time. *Infarcts* may occur and lead to rupture of the heart muscle, or such infarcts may be replaced by fibrous tissue and leave depressed and firm scars. The part affected may bulge from the intracardiac pressure, causing chronic aneurysm, and as the left coronary is most commonly affected such changes are usually in the left ventricle.

Embolism of the smaller divisions of the coronaries is usually found after ulcerative changes in the aortic cusps or at times in pyemia, and the heart muscle contains one or many small abscesses.

When some of the coronary subdivisions are the seat of obliterating arteritis the myocardium presents many areas of fibrous replacement (disseminated fibrous myocarditis).

Cardiac infarcts of large extent are usually found near the apex of the left ventricle, either anterior or posterior, and may involve the septum also; the part is pale yellow, dry and of firm consistence at first, afterward softer, at times laminated with alternate healthy and necrosed muscle. Under the microscope there is absence of striation, granular changes in the muscle cells, loss of staining power, hyaline degeneration in the capillaries, fatty degeneration, perhaps pigment and beginning limitation by fibrous hyperplasia. Such areas of anemic necrosis may dilate or rupture into the pericardium. If the dilatation is rapid without fibrous repair in the neighborhood it is called acute cardiac aneurysm, if slower with hyperplasia it forms chronic cardiac aneurysm; with the latter, especially, thrombi form on the inner surface.

Simple *anemia* of the heart muscle occurs with general anemia from all causes and is often found in late cachexias; fatty degeneration is usually associated. *Hyperemia* is most often a passive condition and found in the right side of the heart, the veins being very prominent under the pericardium and the muscle a deep brownish-red. *Hemorrhage* is usually small in extent, found about abscesses and infarcts, after violent deaths as asphyxia of the new-born or from death by hanging, sometimes in septic diseases and valvular lesions and in the hemorrhagic diathesis and purpura. Circulatory disturbances in the pericardium may be acute congestion connected with pericarditis or with abscess of the myocardium involving the surface, or after sepsis and violent deaths with extreme dyspnea (asphyxia neonatorum). Rupture of cardiac and aortic aneurysms may fill the pericardium with blood, death occurring from pressure upon the heart rather than from hemorrhage, for the membrane is very tough and not elastic and 200–300 cc. of blood within it, whose loss elsewhere would hardly be noticed, will compress the heart and stop its diastole. In general dropsy and pericarditis with effusion a large amount of fluid may collect in the pericardium, but is not so rapidly fatal by compression because of its slower collection and because it does not coagulate.

Inflammation of the Heart.

Cardiac inflammations may be considered as:

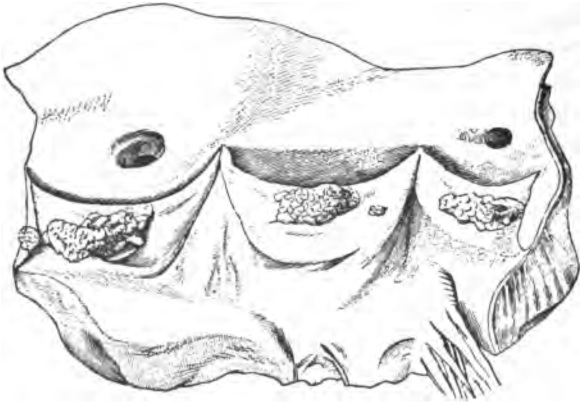
1. Inflammation of the lining membrane, endocarditis
    - verrucosa,
    - ulcerosa,
    - deformans.
2. Inflammation of the muscular tissue, myocarditis
    - purulenta (embolic),
    - parenchymatosa acuta,
    - fibrosa.
3. Inflammation of the enveloping membrane, pericarditis
    - sero-fibrinosa,
    - adhesiva,
    - purulenta,
    - tuberculosa
    - (hemorrhagica).

1. **Endocarditis** occurs most often on the surfaces of the valves, may be acute, chronic or recurrent, and is of two chief forms; the warty or *verrucous*, subacute and recurrent, often the first stage of a valvular lesion; and the *ulcerating* or "diphthe-

ritic," causing rapid destruction of the valve, sometimes developing on a verrucous foundation, sometimes appearing on healthy valves as the result of bacteria (mycotic, malignant endocarditis) in the blood.

Endocarditis Verrucosa.—The seat is usually the mitral or aortic valve, on the ventricular aspect, less often the tricuspid and least the pulmonary; at times also on the parietal endocardium. The vegetations are fine yellowish-white excrescences, often covered with fresh layers of fibrin, and are made up of spindle-shaped cells derived from the endothelium or from the fibrous tissue, with blood plates and their finely granular detritus between them, leucocytes in varying amount and delicate threads of fibrin; into the newer layer of fibrin the connective-tissue cells make their way and thus the vegetation continually increases in size. If they do not coalesce the gross appearance is finely papillary, otherwise there are larger and firmer masses, often ⁻ʳranged on either side of the corpus Arantii and from there

FIG. I.



ENDOCARDITIS VERRUCOSA. (*Ziegler.*)

About the corpus Arantii on each cusp there are fibrous projections and fibrinous deposits on these.

along the lines of contact (lunulæ) of the cusps. The process is thus a combined productive inflammation and thrombosis. If the process is rather more acute the organization is less complete, the elements may degenerate and be washed away and only a scar-like thickening remain; if very acute, as may happen at any recurrence, the valve may be destroyed by round-celled infiltration and necrosis and, if not washed away, such parts may be infiltrated with lime salts and variously distorted.

The most extensive changes of this variety occur in the course of acute articular rheumatism and after it, but they are also found with typhoid fever, measles, scarlatina, tuberculosis, pneumonia, and puerperal sepsis; in certain of these the bacteria corresponding have been found in the vegetations, in others the micro-organism is still unknown in the main disease.

Endocarditis Ulcerosa (Diphtheritica).—Probably the initial step in any endocarditis is a superficial necrosis; in the ulcerating form this necrosis is more pronounced and there is deep and extensive ulceration, starting in the vegetations of a previous verrucous form or appearing first as a series of fine yellowish spots on the endocardium, covered with fibrin and degenerating leucocytes. The lesion progresses as a purulent infiltration or as a rapidly spreading necrosis, and if such dead tissue is carried elsewhere as emboli it reproduces the purulent or the diphtheritic nature of the lesion; these two may occur together. The affected valve may be perforated, or its tendinous cords separated, or large sections may separate and form emboli. The process usually spreads rapidly to the myocardium, especially when the original site is the parietal endocardium, and with this there is almost always suppuration; if the entire thickness of the muscle is involved, purulent pericarditis follows. Emboli occur frequently in the brain (left mid. cerebral artery), the kidney, the spleen, and, if the tricuspid is involved, in the lung. Pyogenic cocci, gonococci, bac. coli communis, bac. pyocyaneus, and others have been found in the lesions of ulcerating endocarditis. The clinical symptoms are usually high fever, profound constitutional disturbance, rapidly fatal course and often some clearly demonstrable infection by microörganisms.

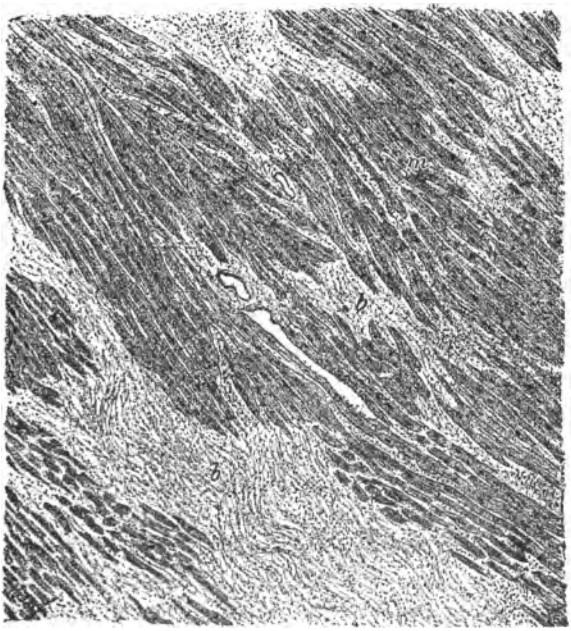
Endocarditis Deformans.—As the result of an acute endocarditis, in old age, and associated with gout and arteriosclerosis, the cardiac valves, especially the aortic, may become thickened, calcified, retracted and stenosed, the cusps of the aortic and the edges of the mitral flaps may be adherent, the chordæ tendineæ of the latter are thickened and retracted, within the aortic cusps there may be large lumps of lime salts under the endocardial layer; the results are loss of elasticity, rigidity and fixation of the valve segments, narrowing of the lumen, *stenosis*, or *insufficiency*, for the valve can neither open wide enough to allow the charge of blood to pass from one chamber to another nor close completely and prevent regurgitation. Embolism from deposited fibrin and broken vegetations, may follow and various disturbances of the circulation, with hypertrophy of the muscular tissue from increased effort, and later dilatation.

Myocarditis is usually secondary to inflammation of either serous covering, or to stenosis and occlusion of cardiac vessels, or to microörganisms and their toxins in the course of severe general infections.

Suppurative myocarditis may follow an ulcerating endocarditis and appear as single or many foci of suppuration throughout the muscular tissues of the heart, the muscle cells undergoing granular degeneration and the intercellular substance tending to loosen about the focus. Such areas of pus formation may follow embolism of the coronary arteries, especially in pyemia, at times also in typhoid and inflammatory rheumatism. Here the lesion begins as small points of necrosis, the vessels containing numerous bacteria as also the affected tissue. The suppurative process follows the intermuscular connective tissue and forms abscesses, and if these open into the cavities of the heart they are converted into aneurysms by the blood forcing its way into them; even complete rupture of the heart may result in this way. The abscess may perforate the septum, or open into the pericardium and set up suppuration there.

Acute parenchymatous myocarditis, sometimes described as diffuse infectious myocarditis, is one of the well-known lesions occurring in typhoid fever, scarlatina, diphtheria and other forms of infectious disease. The heart in such cases is remarkably soft and yielding, the right ventricle is apt to be dilated, and the muscular tissue may present faint yellowish-gray mottlings especially in the portion which works the hardest, the left ventricle. Microscopically such a heart presents a general granular alteration in all of its cells, and in places this has been succeeded by fatty changes also. Macroscopic tears across the muscle are found and similar lesions are seen by the microscope. Inflam-

FIG. 2.



CHRONIC MYOCARDITIS. (*Coplin.*)
m, muscle fibers undergoing atrophy; *b*, fibrous tissue replacing muscle.

matory infiltration of the connective tissue is almost constant if the case has lasted any time, and the vessels of the deeper layers of the pericardium, sometimes of the endocardium, are surrounded by such infiltrate of small round cells; in more severe cases these are found generally through the tissue. Rounded and spindle-shaped fibroblasts occur in numbers and if the case recovers probably cause fibrous scars.

Chronic Myocarditis (Fibrosa).—When many areas of cicatricial tissue are found throughout the myocardium they may be explained as due to changes in the coronary arteries, by which the nutrition of the muscle suffers and its cells are replaced by fibrous tissue, or as the remains of infarcts; a process of this nature may be described as an arteriosclerotic myocarditis and is fairly common as a senile change in the later years of life. But a similar lesion may occur apart from changes in the vessels, in connection with valvular diseases and hypertrophy, and accompanying syphilis and chronic alcoholism. In such cases there are numerous very fine spots and lines of new connective tissue distributed through the myocardium, making the muscular bundles more prominent by contrast, and the consistency of the organ may be much harder than normal. If degenerative changes have accompanied the process the color is mottled with gray and yellow, perhaps best seen on the inner surface of the left ventricle just below the aortic valve and in the papillary muscles; or the entire myocardium is brownish from the pigment derived from the degenerated cells. Microscopically different portions present various stages of the process, from simple and localized infiltration with round cells to relatively large bands of connective tissue replacing and constricting the atrophied muscle bundles. The lesions may be chiefly about the vessels or apart from their distribution and with degeneration the cells look granular or fatty. The nuclei of the muscle cells are apt to be distorted or swollen. Hyaline changes may be present, and as a rule degeneration and fibrous replacement go on together.

Pericarditis.—The sero-fibrinous form of this disease usually occurs in general infections but may also follow extension from neighboring parts, pleurisy, myocarditis, etc. When pericarditis occurs without general infection, as a single disease, it may be called idiopathic, but even in such cases is to be explained by the microörganisms discovered in the exudate. The disease is characterized by the occurrence of serum in the pericardium in large quantity, and this exudate may be the first and chief lesion or may precede or follow the production of fibrin on the pericardial layers. Clinically the friction murmurs of the latter condition may often be noted, then disappear as the fluid accumulates to reappear as it is absorbed. The fibrin is most copious over the heart rather than over the parietal layer of the membrane, beginning at the root of the great vessels as a fine and delicate gray or yellowish membrane, which may at first be easily lifted from the serous surface. During the heart's motion the two layers of the pericardium move so that different areas are in apposition at different times, hence the fibrinous bridges thrown across from one layer to the other are drawn out and frequently broken apart, with the resulting formation of little tags and tassels (cor villosum). In time the fibrin turns a deeper yellow and becomes more brittle; under it the serous membrane will be found to have lost its smooth and glistening look, appearing more as ground glass which does not permit one to recognize the tissue beneath, and the vessels may be prominent from the inflammatory hyperemia. Under the microscope these vessels are distended, in places irregularly, and surrounded by small round cells; the cells of the serous surface are either swollen and pushed apart by the round cells or entirely lost and at times the relation of the fibrinous exudate to the vessels is very clear. From the connective tissue of the epicardial layer a new growth of fibroblasts takes its point of departure, organizing the layer of inflammatory products and carrying the new vessels up into it; from these it is common to have hemorrhages.

Adhesive pericarditis (sicca) differs from the preceding chiefly in the absence of any great amount of fluid exudate.

The two layers of the pericardium are early united by fibrin and these recent and tender adhesions become organized and firm. A similar result may attend the first form after absorption of the fluid. In many cases the two layers do not adhere but the site of the fibrin, which becomes organized, is marked by opaque whitish spots on especially the visceral layer ("milk spots"). Even when the two layers appear to be firmly and wholly united, careful search will discover areas where a lumen persists, still clothed with endothelia. In severe degrees of obliteration of the pericardial sac, especially when there are recurrent attacks of inflammation, the myocardium beneath the serous layer may undergo marked fatty change.

Tubercular Pericarditis.—From an infected pleura, from caseous lymph nodes of the mediastinum and less often from tubercle of the heart wall, the pericardium may become the seat of a typical tubercular inflammation, usually of very chronic course and of the fibrinous type. Pericarditis in connection with other diseases (acute rheumatism) may become infected with the tubercle bacillus later. From the vessels of the imperfectly organized fibrin hemorrhages are frequent. In the exudate the usual elements of tubercular processes are discovered, caseous areas with small lymphoid cells and giant cells and tubercle bacilli.

Purulent pericarditis usually follows suppurations in adjacent organs, purulent pleurisy, abscess of the myocardium, perforation of gastric ulcer upward, severe peritonitis carried through the diaphragm, esophageal neoplasms, and pyemic infections. Otherwise it may begin as any inflammation of the pericardium and the exudate becomes abundant and purulent, the myocardium is at once and powerfully affected, becoming edematous and infiltrated with pus in its outer layers, and usually the lesion is rapidly fatal. The collection of pus is often not very large but it may reach such a degree as to distend the cavity and finally perforate into the pleura of either side or elsewhere.

Hemorrhagic pericarditis is simply one of the foregoing with relatively copious bleeding from the newly formed vessels, or in the course of some general disease (purpura, scorbutus) associated with frequent hemorrhage.

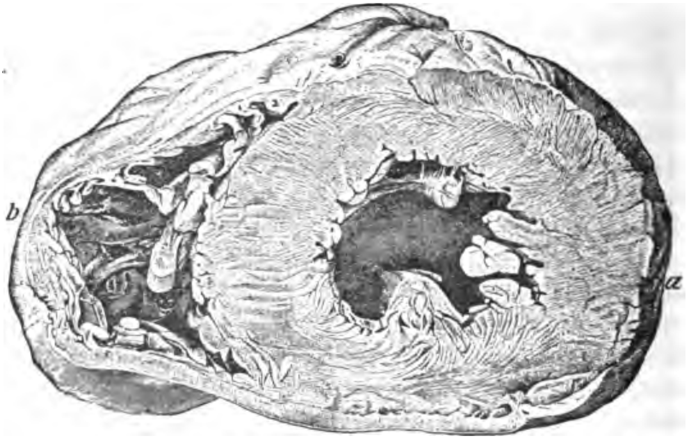
Cardiac Hypertrophy and Dilatation.

Three forms of cardiac hypertrophy are sometimes mentioned, the *simple hypertrophy* where one or more of the chambers have thicker walls than normally without suffering any change in capacity, the *excentric*, where the hypertrophy coexists with dilatation, and the *concentric* where the wall is thickened and the cavity diminished; the latter is really a mistaken interpretation of a hypertrophied heart which has stopped in systole and should not be considered as a distinct form.

Cardiac hypertrophy may accompany changes in the valves or the systemic vessels, or occur idiopathically. In the latter case the name simply means that there is no apparent explanation in the vascular system or elsewhere for the increase in size, and such hypertrophy is seen in the heart of those who habitually consume large quantities of beer, in those who have led an especially athletic life, and in those of nervous habit who have through life been afflicted with palpitation of the heart following every slight bodily and emotional effort. In all these cases the heart may later in life give evidence of being tired out, a presenility due to its excessive functional activity, and in some cases there may be degeneration of the muscle and dilatation of the cavities also. A "normal" size of the heart is difficult to give as it depends so much upon the bodily conformation and the muscular development and activity of the individual, but it bears a certain proportion to the height and a more important one to the weight; being of a capacity of 150–190 cc. for every 100 cm. of height (hypertrophied hearts reaching 300–400 cc.), and weighing 300–350 gm. for the male and 240–250 for the female, all these figures being for the adult. (See Pt. I., p. 312.)

Hypertrophy associated with other diseases may be found in cardiac, vascular and renal affections especially, and in most of the cases is a result of increased work comparable to any of the voluntary muscles which grow by exercise. From one point of view the heart and the arteries form but one continuous organ, every beat of the former running along the latter to the farthest point where there are circular muscular fibers, and these both continue and lessen the work of the heart. Any change in the vessel walls which impairs their contractility in the same proportion increases the work of the heart, for it can no longer leave to the vaso-motor mechanism the work of carrying the blood wave to distant parts. If in addition the walls of many of the vessels are stiffened, so that friction increases with loss of elasticity, the heart's work is so much the greater, and the part which feels this most constantly, the left ventricle, displays the greatest increase in size. This is the explanation of the constant hypertrophy of the left ventricle found in renal diseases, for with them there is associated, often over long periods of time, a vascular rigidity throughout the body which enormously multiplies

FIG. 3.



HYPERTROPHY OF LEFT VENTRICLE. (*Ziegler.*)

a, hypertrophied wall of the left ventricle; *b*, wall of the right ventricle. The intraventricular septum also is thickened.

the effort of the left ventricle to send the arterial stream through the system. Hence without valvular changes cardiac hypertrophy and nephritis are commonly associated. A similar explanation suggests itself for cases where there is general narrowing of many of the vessels, as in senile atheroma, and also where there are dilatations of the larger arteries (aneurysms). Perhaps the pressure exerted upon the abdominal vessels during the latter months of pregnancy will explain the lighter degrees of the same change commonly found in that condition. When the hypertrophy is marked it is termed "cor bovinum." These systemic causes chiefly affect the left ventricle, but similar increase of work for the right side of the heart will give a corresponding hypertrophy of its walls. Very commonly in emphysema, because of the general atrophy of the pulmonary capillaries and consequent increased resistance to the entrance of blood into the lung, the right ventricle shows a high degree of hypertrophy, and similar changes follow chronic pleurisy, fibrous induration and other lung conditions.

In consequence of valvular lesions hypertrophy of the heart is common, and this may follow either stenosis or insufficiency of any orifice or combinations of the same ; nearly every stenosis means also an insufficiency of the same valve, but not every insufficience a stenosis. Valvular lesions work in two ways, the arterial side containing too little blood with each systole and the venous too much, whether the lesions be narrowing or insufficience or both. This will be seen if followed out for any one valve and can then be applied to any other. Thus if the mitral be insufficient, at every contraction of the left ventricle some of the blood which ought all to go out of the aortic valve will be driven in the wrong direction back through the mitral. Consequently the left auricle contains too much blood and will be distended, and the effects of this, as the blood is practically incompressible, are almost like force transmitted through a solid body back through the pulmonary veins into the lung capillaries and later on through these into the right ventricle. At the same time the ventricle, because of the loss of some of its blood prematurely and abnormally through the mitral, has too little to contract upon, the muscular effort is carried on at a great mechanical disadvantage, and the aorta receives too small a charge, and that is abnormally and irregularly delivered. With the diminished supply to the aorta, all the great vessels and the arterial system in general contain too little blood while the venous system as a whole is overfilled.

If, on the other hand, the mitral is stenosed, while its insufficience is not yet marked, the ventricle receives in diastole too small a charge of blood, the auricle is constantly overfilled though not subjected to the sudden bursts of the returning column of insufficience, the arterial tree is improperly supplied, and again the tendency is toward venous engorgement and stasis. When both defects are present the disturbance in pulmonary and general circulation must of course be far greater.

Increased work being given to the heart in the above conditions, severe and fatal consequences are delayed only by its ability to meet the added calls for a time by increasing the muscular size and power of the cavity walls most involved, and such *compensatory hypertrophy* is most commonly noted in the ventricles, especially the left. Such compensation is best attained when the patient has a good general bodily condition and when the valve defect is fairly slow in its development, and so long as the myocardium is normal it is the usual result. The compensation has limits which are sooner or later attained and then the hypertrophied muscle ceases to increase still further in size and power, and afterward it passively yields to the internal pressure caused by the obstacle to the circulation and the cavity dilates. With this there is a rapid and marked loss of power in the heart and a notable increase in the gravity of the symptoms.

An acute dilatation, usually temporary, may follow extreme muscular exertion. With constant severe exercise there may be permanent dilatation with or without hypertrophy.

Valve lesions of the left side of the heart are often associated with changes in the lung from the chronic passive congestion produced (brown induration, pneumonia of heart disease, chronic bronchitis). From the right side, if the blood is obstructed in its passage into the lung, it backs up into the venæ cavæ, and aided by gravity causes passive congestion of the liver, at times very marked, with atrophy of the cells and fat deposit (nutmeg liver), and various edematous changes are noted in dependent portions (feet and legs). The gross appearance of the heart varies according to the part involved; if the right ventricle is chiefly enlarged it forms more of the apex than normally and gives a blunt and rounded look to this part; if the left ventricle is chiefly hypertrophied, it presents far more than normally when the pericardium is first opened, the apex is displaced downward and to the left. The organ is heavy in either case but the weight is specially great when the left side is enlarged (cor bovinum, up to 700–800 gm.); in either case the auricles also may be increased in capacity and the walls thickened. The hypertrophied heart is often firmer than normal; if dilated, and especially with fatty or other degeneration, it may be less firm.

The stenosed mitral may resemble a firm white truncate cone projecting into the ventricle, just admitting the tip of the finger or not even so large an object, dense and rigid, the chordæ tendineæ opaque, white and shortened. The insufficient valve admits two or three fingers and perhaps they may be separated a little while in the opening, the valve flaps are thick, retracted and distorted, and the row of interdigitating nodules along their edges lost in the thickened tissue; the thin fold of endocardium beyond these nodules which normally completes and ensures the closure of the valve is early retracted and lost. The flaps may also be the seat of calcareous deposits and the papillary muscles show fatty or fibrous changes. On the aortic valve the cusps are thickened, retracted, rigid, infiltrated by lime salts, mutually adherent over a larger or smaller area; both mitral and aortic may be roughened with verrucous growths and recent fibrin. Similar changes occur to a less degree on tricuspid and pulmonary, the last least often affected. With such valve changes the myocardium seldom escapes, various degenerative changes being present, most apparent when the coronary arteries are also diseased.

Cardiac hypertrophy is in great part a true enlargement of the individual muscle fibers, with enlarged nuclei also; the connective tissue does not increase in the same proportion and hyperplasia, simple numerical increase of normal muscle cells, plays the least important part.

Cardiac Atrophy and Degeneration.

Atrophy seldom affects the endocardium except as connected with scars and other results of valvular endocarditis and myocarditis; in the valves small fenestra may be formed in such conditions, but these are almost without significance.

Degenerative changes in the muscular tissue of the heart are of great importance, caused by either local or general disorders.

Simple atrophy, or *brown atrophy*, is the result of long-standing processes which affect the muscular cells; sometimes appearing as a senile change in the very old, sometimes even in early years after long and exhausting disease; in a circumscribed form it may occur in a single ventricle which has first suffered hypertrophy, or about tumors and after myocarditis. Even marked degrees of this degeneration may give no clinical symptoms. Microscopically the individual fibers are noticeably slender and contain about the nuclei and in the site of atrophied cells numerous fine grains of yellow-brown pigment; where the sheath of the fiber remains it appears as a tube nearly filled with such granules. The pigment is derived from the muscle substance and contains no iron.

An apparent atrophy sometimes may be found in the case of delicate women whose muscular tissues throughout are ill developed, or as part of a general aplasia affecting the circulatory organs.

Fatty changes in the myocardium may present themselves as an increase of the subpericardial fat. This is normally found at the base of the organ and along the vessels, but may nearly cover the entire heart and on section be found in the wall even to the endocardium. Such a condition is a replacement of degenerated muscle by fatty tissue, a *fat invasion*, and is a common change in the hearts of beer drinkers and obese anemic women. Such hearts are incapable of much added effort and, if marked in degree, explain some cases of sudden death after muscular and emotional strains.

Fatty degeneration of the single muscle fibers may follow local mechanical interference with the blood supply, from pressure upon the vessels by pericardial exudate or thickening of the coronary vessels; or it may be the result of general conditions, as chronic alcoholism, phosphorus poisoning; or it may follow acute infectious disease, as scarlet, variola, diphtheria and especially typhoid fever. Certain disorders of the blood may be the exciting agent, and thus it is common in pernicious and other marked anemias and in the cachexias of syphilis, tuberculosis, nephritis.

In the gross the left ventricle shows fine mottlings of a yellowish tinge under the endocardium and when this is limited in degree perhaps the papillary muscles are the site of most evident change; the tissue is soft, friable; the cavities are larger than normal and the heart has stopped in diastole. In very marked fatty change such appearances are repeated in both ventricles and to a less extent in the auricles; at its highest, the knife may even be streaked with a yellowish smear of fat after section of the myocardium. Fatty changes in the heart muscle are very easy to see microscopically, but slight degrees may entirely escape gross examination. Under the microscope a bit of fresh tissue shows loss of striation, though this may persist in many cells, but the single muscle cell is here and there converted into a mass of very finely divided fat granules, which in other places join to form large drops; these may be arranged in lines following the normal course of the cells. Albuminous degeneration may coexist, which clears with the addition of acetic acid.

Other degenerations may occur in the myocardium, *amyloia* changes in the endocardium and in the vessels, *hyaline* degeneration of the connective tissue, *calcareous* deposits in thickened valves and papillary muscles, but as a general rule these are relatively of little importance.

Various degenerative lesions have been described in the cardiac ganglia, after sudden death in chloroform narcosis and from angina pectoris, either as granular changes in the ganglia with fat formation or as of an inflammatory nature. From their development the cardiac ganglia belong to the sympathetic system and even if these degenerations are not the result of methods employed, their interpretation remains a matter of uncertainty.

Wounds of the Heart.

Penetrating wounds may result from stabs, from bits of broken rib or from foreign bodies in the esophagus; if their main axis runs across the muscle fibers, gaping is marked and blood fills the pericardium at once; running parallel with the fibers and of slight extent they may not permit much bleeding and may heal; foreign bodies, like bullets, may even become encapsuled in the myocardium. Similar wounds are usually found in the corresponding part of the pericardium. One case is on record where a heavy load of wood coming down a hill pinned a man against a tree trunk and the heart was found torn from its great vessels and lying free in the pericardium; there was no external wound. In conditions of marked fatty and other degeneration the heart muscle may rupture, and one form of this is fairly common as accompaniment of the death agony when strong convulsions mark the close of life. Other cases follow myocarditis with abscess, tumor of the heart wall and similar affections. The tears are usually in the left ventricle and disposed across the fibers or in the basement substance. Similarly a cardiac valve may be torn partially or completely, and those of the left side more frequently involved than the others.

Tumors of the Heart.

Round-celled sarcoma may develop from the connective tissue of the myocardium and occasionally portions of tumor which have been carried in the venous circulation may settle on the endocardium and persist. Cases of lipoma, fibroma, myxoma and a few others have been reported; carcinoma of the breast may pass to the pericardium and the heart itself by contiguity. Almost all such tumors are secondary. Tubercular and syphilitic granulomata are not uncommon in severe cases of these maladies.

II. DISEASES OF THE VESSELS. ARTERIES.

Inflammation.—Acute inflammation of an artery (*arteritis*) usually follows the entrance of foreign bodies into its lumen, either infectious or otherwise, and affects the intima first; the endothelial cells become swollen and their attachment loosened, pus cells appear in the tissue of the wall from the vasa vasorum, the inflammation extends deeper to the media or even to and through the adventitia. *Periarteritis* may follow injuries from without or extension from within, and is usually exudative; the vessel at first contracts and lessens its lumen, then as the muscular elements in the media become edematous the vessel dilates. Degenerative changes of part or the whole of the vessel wall follow, thrombi are common; the latter may organize and bring the process to an end or soften and cause embolism of vessels beyond.

Chronic arteritis has many names, arteriosclerosis, endarteritis deformans, atheroma and others, and consists in a series of new tissue formation and subsequent or contemporary degeneration; the former affects specially the deeper coats of the vessel, the latter the intima. Such a chronic arteritis may be either diffuse or localized, and shows a tendency to affect certain vessels more than others, as the aorta, the vessels at the base of the brain, the splenic artery. At times a few of the main trunks are diseased, at times only the smaller divisions, or it may be a general process.

Diffuse arteriosclerosis may affect only the arch of the aorta, its commonest seat, or the entire length of thoracic and abdominal portions. From the gross appearance of small roundish or oval plates of thickened and degenerated tissue it has received the name *arteritis nodosa.* These nodules are but little if any prominent above the general surface of the intima and seem at first to be mere connective-tissue thickenings, and the essence of the process is a replacement fibrosis following changes in the vasa vasorum. These interfere with the elasticity of the media, because of disturbed nutrition, new connective tissue is formed, not being so elastic as the tissue replaced this new connective tissue permits a distention of the media and its own nutrition suffers; closely connected with this come degenerative changes, often hyaline, sometimes fatty and mucoid. At the same time there is a deposit of calcium salts in the (hyaline) degenerated tissue and such a place projects into the lumen of the vessel and feels of stony hardness. Deeper in the tissue there are little foci of degeneration presenting cell detritus, fat granules, cholesterin tables and salts of lime, the whole being semifluid and resembling the contents of sebaceous tumors. This circumstance has given the name *atheroma* (gruel-like tumor) to the process. The endothelial cells of the intima in such a place are early lost and the intima suffers fatty degeneration, the result is an opening into the vessel which permits the softened material to be swept away by the blood stream and the remaining cavity is called an *atheromatous ulcer*, though it has but little in common with typical ulcers. Coagulation of fibrin at such a place forms white thrombi and these also contain calcium salts at times. The vessels of the wall are often surrounded by signs of productive inflammation; they may also be increased in number. Fibroblasts and occasionally giant cells are found and a scar may replace the ulcer after its contents have been swept away; or the new formed tissue may degenerate in its turn and make the injury worse.

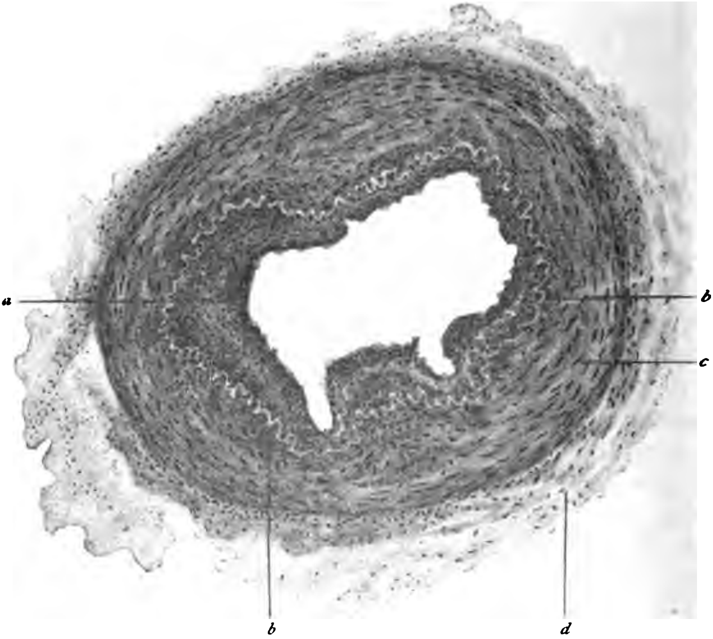
The results of such a process vary with the seat and degree, but if marked there is such an increase in the intravascular friction that the left ventricle hypertrophies; the vessel itself is lengthened and dilated if large, its lumen is usually decreased if small. The thickened intima is relatively greater and the cardiac impulse is less marked and the muscular elements retain their contractility for a time; thus the small artery becomes a tortuous stony tube in a contracted condition. From such a stenosis the parts beyond must suffer in their nutrition and in this way senile gangrene may be explained, as well as softening of the brain. If the organs are involved, as the kidney, they become smaller and lose their function. Embolism from fibrin deposits are always possible.

The general tendency in old people for calcium salts to deposit in all tissues, together with decreasing nutrition, explain arteriosclerosis as a senile change; but in addition there are other factors, abuse of alcohol, venereal excess, syphilis, excessive muscular activity, which enter into its production; gout, lead-poisoning and other forms of intoxication also lead to such changes in the vessels.

As a localized lesion chronic arteritis may accompany any productive inflammation (*arteritis obliterans*) which passes over from another tissue and involves the vessel walls. This is common in chronic nephritis, emphysema and tuberculosis of the lungs, and in certain lesions of the central nervous system. Certain diseases are characterized by a general tendency to excite new connective-tissue formation throughout the body and of these syphilis and tuberculosis are typical; in the latter case the peculiar structure of the tubercle and the presence of the bacilli are distinctive, but syphilitic lesions are less clearly demonstrated. Gumma may occur in the walls of vessels as elsewhere, starting in them or in the neighborhood. Such lesions have been found in the cerebral vessels, in the carotids and in the portal vein; and they differ from atheroma in being confined to one or a few vessels, having less calcium salts deposited, often closing the lumen entirely, and presenting a tough yellowish thickening of either the intima alone or the whole arterial wall. The vessel may be dilated proximal to such a gumma; in the brain, where the clinical symptoms are most pronounced, softening is a common result. Tubercular lesions are commoner in the veins than in the arteries and will be mentioned in that connection.

Various forms of *degeneration* may be noticed in the arterial walls. Hyaline changes affecting the whole wall are common in various organs with inflammatory processes, as in the glomeruli with nephritis, also in the walls of aneurysms, in vessels closed by thrombi, and it is often the first stage of arteriosclerosis. It appears as a homogeneous and structureless substance replacing the elements of the vessel wall. Fatty degeneration occurs in

FIG. 4.



ENDARTERITIS OBLITERANS. (*Coplin.*)

a, intima thickened and thrown into folds; *b*, elastic coat; *c*, media, irregularly thickened; *d*, adventitia.

both intima and media, with chronic arteritis and independently, appearing in the gross as fine yellowish dots and lines under an endothelium which is clouded. Microscopically the fat granules and drops are found in the cells, muscle and other, and afterward between them also. From the internal pressure such a weakened spot yields slowly, forming an aneurysm, or suddenly after great increase of the stress. Deposit of lime salts in the arterial walls accompanies certain chronic bone disease with absorption of the lime, the material being laid down again in the vessels. Sometimes it appears without evident cause, especially in the arteries of the uterus, the abdomen and the legs. Amyloid degeneration especially affects arteries, and of them particularly the smaller divisions; by treatment with iodine they appear as brownish twigs and branches.

Aneurysm.—Dilatation of arteries in which one or more of the coats are involved are called *aneurysms;* if a wound of the vessel has allowed the blood to escape into surrounding tissues and it is confined there it may be termed *aneurysma spurium* or *false aneurysm*, and in the recent state this is diffuse; in older and encapsuled collections it is circumscribed; in the latter case the vessel may still communicate with the hematoma and then the resemblance to true aneurysm is very close.

Dissecting aneurysm is the name given to vascular dilatations which have arisen from tears in one coat (intima) or occasionally in two (media also), which allow the blood to force a path along the vessel between its layers, making fusiform swellings along a portion of the artery. Such a collection may force its way out into the tissues or back again into the vessel. Dissecting aneurysm is most common in the large vessels and perforates in the direction of least resistance, into the pericardium frequently. In the brain the blood may pass out of the vessel entirely but be confined within the periarterial lymph channel. Dissecting aneurysm is most commonly caused by violence, but may occasionally follow disease and degeneration in the vessel wall.

True aneurysm is the local dilatation of an artery in consequence of weakened resistance in its walls, the blood-pressure forcing the weaker part outward; either at first or as a result of the stretching, there is a solution of continuity in the intima, then in the media, less often in the adventitia. The commonest form of true aneurysm is the *sacculated*, where the dilatation is lateral, spheroid, not involving the entire circumference of the vessel; often seen on arteries which are arched and then on the convex aspect. Other forms are the *diffuse*, a relatively long section (entire thoracic aorta) being uniformly dilated, and the *fusiform*, where a shorter length is involved and the spindle shape is more evident. Combinations of one or more are common.

Once formed, the aneurysm may persist for a long time with but little change. Internally thrombi slowly form in layers, soft parts in the neighborhood accommodate themselves to the pressure; hard structures, as bone, are slowly eroded by the steady pressure, probably because the periosteum atrophies at that point. The continuous deposit of layers of thrombus may lead to healing of the aneurysm if the opening into the vessel is small and the adjacent wall fairly healthy. Otherwise, in spite of the stratified clots, the aneurysm bursts and a hematoma forms in the tissues, or a cavity is filled by the rapid bleeding with fatal consequences.

Aneurysma verum has sometimes been termed spontaneous, but this adjective can have only the signification of non-traumatic in such a case, for the dilatation is always preceded by inflammatory or degenerative changes or both in the vessel wall and a healthy artery cannot dilate spontaneously enough to form an aneurysm. The essential feature of any such lesion of the artery, leading to formation of an aneurysm, is that the elasticity of the media is decreased and consequently its resistance. A common explanation of aneurysm from atheroma, making a solution of the intima and allowing the blood to exert pressure on a (relatively) healthy medium and thus dilate the vessel, cannot be admitted as probable, otherwise aneurysm from nodose atheroma of the aorta would be a more frequent post-mortem find. The common appearance of atheroma without aneurysm and of aneurysm without atheroma, refers the production of the dilatation to some less evident etiology. Perhaps it is closely connected with congenital imperfections in the development of elastic tissue generally, and a side light is thrown upon the subject by the occurrence of emphysema in certain families; here the elastic tissue of the lung is at fault, and if the influence of heredity may be admitted to explain its causation, perhaps a similar hypothesis will hold good for aneurysm. Aneurysm is commoner in men than in women, in certain countries (as England) more than in others, and in the ten years from 35 to 45. Although the chief danger of an arterial dilatation, namely its rupture, usually coincides with some great muscular effort and consequent increase of intravascular pressure, it is interesting to note that quite frequently aneurysms break while the individual is asleep; the medico-legal importance of this may be critical.

If the wall of the aneurysm is formed by very slow dilatation it may consist of all three coats, but in most cases the elastic and muscular elements are entirely absent and sooner or later the intima also. One form of multiple congenital dilatations is common about the bifurcation of small vessels, and here the elastic tissue is wholly lacking while the muscle cells are few and imperfectly developed. A form of rapidly developed aneurysm depends upon suppurative and other changes in the vessel wall, derived at times from the neighborhood, at times from septic emboli; the emboli are occasionally parasites or fragments of them, eggs, etc. A form of multiple miliary aneurysm occurs in the brain and leads to cerebral hemorrhage, especially affecting the smallest arteries and of these the branches of the lenticulo-striate most commonly; they are due to degenerative changes in the media, as elsewhere, though often associated with atheromatous and vaso-motor lesions.

The large vessels vary in their disposition to aneurysm, the thoracic aorta being by far the commonest seat, followed by the popliteal, the femoral, the abdominal aorta, the carotids, the subclavian, etc., in the order of frequency.

New Growths.—Hypertrophy of large sections of the arterial tree may accompany similar change in the heart. Tumors formed of new and dilated arteries occur and similar elements are found in many other neoplasms. Tumors may attack the artery from without, involving the adventitia, but seldom the media; at times the endothelium proliferates even though the media remains uninfluenced by such an attack, giving rise to endothelioma and combinations. Or the vessel may become the seat of a neoplasm from embolism of small portions of the new growth, the endothelium growing into and continuous with the embolism or replaced by its cells. As a general rule the vascular wall exhibits a high degree of resistance against tumors growing in the neighborhood. New vessels formed in tumors seldom reproduce the normal type of structure, especially in sarcoma, where the blood circulates through simple channels in the neoplasm.

Veins.

Thrombosis.—The coagulation of the blood within the cavity of a vein may occur from many causes and vary in results according to the size of the vein, its anatomical site and relations, and the septic or aseptic nature of the thrombus. General aspects of the subjects have been dealt with in the first volume (see Vol. I., p. 40). Special features of individual veins may be inferred from their relations and the ease of establishment of collateral circulation.

Inflammation.—*Phlebitis* is described as peri-, meso-, or endophlebitis according to the position of the inflammatory process in the wall of the vessel; if caused by inflammation in adjacent tissue it usually begins as periphlebitis, if from a septic agent within, as an endophlebitis. It may be an acute infectious inflammation, frequently of a superficial vein and following injury. The anatomical steps are first a round-celled infiltration of the adventitia which may go on to be purulent, this process following the course of the vein and making smaller or larger abscesses, combined with thrombosis of the contained blood as soon as the wall of the vein is severely involved. Such thrombus formation for a time prevents the access of pus and pyogenic organisms to the general circulation, but at last the thombus also is destroyed and the infectious material is able to enter freely. With the latter, multiple septic embolism is the natural result: if such entrance is prevented and the course of the inflammation is favorable the thrombus may organize and the lesion heal.

Suppuration in the course of the portal vein is called *pylephlebitis* and follows pyogenic infection of any of the general or hepatic branches of the vein. Thrombosis and purulent softening occur, the vein in a great number of its branches is filled with a puriform fluid, such branching abscesses reach all through the liver; the inflammation may pass from the vessel to other tissues. Capillary stasis and thrombosis occur throughout the territory of the branches of origin with hyperemia and loss of function in the organs involved. The disease may be rapidly fatal or last for a week or more.

Phlebitis occurs also in chronic forms and is sometimes called *phlebosclerosis* because of its resemblance to arterial sclerosis. Slight and localized forms may be found in connection with dilated veins and slowed blood current in varix generally, the veins being increased in thickness and distorted. As a general condition it may accompany arterial sclerosis or rarely occur by itself; the veins of the lower extremity are specially liable to such thickening and show in the gross small scattered areas of denser tissue, due to round-cell infiltration, hyaline degeneration and productive change of all the elements of the wall. At times there are no gross evidences though the microscope reveals those mentioned.

Varix.—Dilatation of a vein may affect the vessel just above a valve, which then may become incompetent or torn off, or it may involve a long stretch of the vessel, tapering to each end and giving the fusiform phlebectasia, or in more marked cases the vessel is elongated and very tortuous (cirsoid form), or on the side of the vessel, similar in form to the sacculated aneurysm, there are few or many protrusions; these may press against each other and become continuous by atrophy of the walls at the point of greatest pressure, so that the appearance of erectile or cavernous tissue is reproduced. Thrombi may form in such dilated spots and by the deposit of lime salts become converted into phleboliths. Such a process is common in old age in certain venous plexuses, as the prostatic. Periphlebitis may follow or the phlebolith, which acts as a foreign body, may become encapsuled and separated from the vessel in which it was formed. The section whose circulation is interfered with as the result of such varices and phlebectasias will suffer in varying degrees according to circumstances: usually edema is marked, a mucous surface is the seat of chronic catarrh, a skin surface as on the legs becomes the seat of chronic ulceration with eczematous and other complications. These varicose ulcers heal with difficulty on account of the imperfect nutrition of the part, and break down again readily; pigmentation is common. The skin may also be the seat of productive fibrosis and be many times thicker than normal, its surface creased and folded or warty and cracked, with passive serous exudation. The clinical appearance resembles elephantiasis and is often complicated with eczema and ulceration. The floor of varicose ulcers is apt to become very hard and cicatricial. The veins may be opened by ulceration and severe bleeding result. The two main factors are mechanical interference with the return flow of blood and pathological changes in the venous walls; the former is seen in parts where physiologically a long column of blood tends to dilate the veins passively by its weight, as in the scrotum and on the legs, with the results of pressure in the abdomen from enlarged uterus, chronic constipation, and the increased resistance to the venous current resulting from emphysema and cirrhosis. Disease of the vessel wall is almost constant; some form of sclerosis which lessens its resisting powers. Certain forms of dilated veins have received special names, *varicocele* if the spermatic veins are dilated, *hemorrhoids* if those about the lower end of the rectum, *caput medusæ* if the superficial abdominal veins dilate to relieve the obstacle in the portal vein caused by atrophic cirrhosis and tumors of the liver. Phlebosclerosis and varix usually co-exist, but either may be found apart from the other.

Neoplasms.—Primary tumors of the veins, as of the arteries, are among the pathological rarities. Secondarily they may be involved by any tumor, especially by malignant forms, and the veins possess less resisting power than the arteries. Local dilatations, or tumor masses formed of such dilated veins, are common in the liver under the name of angioma. Infectious granulomata may affect veins, especially tubercle, and such a hidden source for rapidly generalized tuberculosis must always be kept in mind. Thus a superficial vein may be involved from a caseous lymph node, and such venous tubercles are very common in the vessels of the lung. Syphilis may appear congenitally in the portal and other veins, and later in life gummata occur here as elsewhere.

Lymphatic Vessels.—Inflammation of these vessels is called *lymphangitis* and may be either simple or septic. It follows superficial wounds with loss of substance and presents itself as a red line of hyperemia along the track of the vessels involved, progressing till the nearest node is reached, which then swells and becomes tender. If septic, the node is apt to suppurate and form an abscess and smaller abscesses may be formed along the course of the vessel. The organisms recovered from such inflammations are the pyogenic cocci, aureus, epidermidis albus and others. Severer grades of such inflammation lead to the development of large phlegmonous abscesses and regularly occur with serious post-mortem and dissection wounds; such lymphangitis is especially apt to follow injuries received at autopsies made very soon after death, in cases of infectious disease, puerperal fever, etc. At times it begins as an infection of the lanugo hair-follicles (sycosis) and so passes through the unwounded skin.

Dilatation of the lymph-vessels is not so common as in the blood-vessels. The causation resembles that of varix, an obstacle to the centripetal current passively blocking and dilating parts more distally placed; previous weakening of the wall by inflammation plays a part here also. Such lymphangiectasis is common in elephantiasis Arabum (see Pt. I., p. 235). Tumors and scars which lessen the lumen of a chief lymph-vessel (as the thoracic duct) may produce lymph stasis distally, and through any defect in the surface there may be an outpouring of lymphatic fluid (lymphorrhagia). This is usually white and opaque, like milk, and contains many fine granules of fat and a few white cells. Certain parts especially are apt to be the seat of lymph stasis, with thickening of their elements or hypertrophy and exudation of lymph; among these the scrotum, labia and thigh may be mentioned.

The relations of the lymph channels to tumors is important in two ways. Tumors formed of newly developed lymph-vessels are called lymphangiomata,—simplex when they are not very much enlarged in caliber, cavernosum when dilated; the latter form is usually a congenital condition affecting especially the upper end of the alimentary tract (macroglossia). Lymphangioma cysticum is a similar tumor with very wide lymph-vessels and occurs at times about the neck and the sacral region; the contents of the cysts is usually very fatty, at times partly coagulated. Certain forms of endothelioma begin in the lymph spaces of various tissues, often on serous surfaces. The second important relation of lymph-vessels is as the way along which malignant tumors spread, particularly carcinoma; this is a fact of the utmost practical importance clinically, in all operative procedures on malignant tumors (Pt. I., p. 171).

Infectious granulomata regularly involve the lymph channels, tuberculosis especially, as seen in tubercular ulcers of the intestine; and syphilis, in nearly all of its stages, affects the superficial and deep nodes. Doubtless both diseases spread frequently along lymph channels.

The Blood.

Physiology and Morphology.—Specific gravity: normal 1.054–1.060. It is increased with muscular activity, menstruation, high altitudes, skin lesions, and in the new-born; decreased in chlorosis, simple and pernicious anemia, leukemia, and with malignant tumors.

Red Cells.—Average diameter 7–8 μ; achromatic while living, acidophile when fixed; considered as vesicles containing semi-fluid contents or as viscous solid masses with an elastic stroma which is condensed peripherally. Variations in size include: *Microcytes* from 4 to 1 μ in diameter, in chlorosis and pernicious anemia, with deficient hemoglobin. *Megalocytes*, 10–20 μ, hemoglobin increased or diminished, in severe grades of anemia. *Poikilocytes*, various pear-shaped and distorted cells, in severe anemia with degenerative changes in the red cells. Nucleated red cells occur as *normoblasts*, 7–9 μ, in various anemias; their presence regarded as reparative; *megaloblasts*, 10–20 μ, the nuclei single or multiple, may show mitosis and fragmentation (karyorhexis). Over 20 μ in diameter they are called *gigantoblasts*. Presence indicates pathological cell formation in the marrow, occur in primary and secondary pernicious anemia.

Number: 5 million in males, 4 million and a half in females, in the cubic millimeter; varies with time of day, digestion, sex, menstruation, pregnancy, and nervous disorders; also increased (relatively) in profuse discharges from bowels, endocarditis and venous stasis, phosphorus and CO poisoning. Condition termed *polycythemia*. In hemorrhages the number diminishes: *oligocythemia*.

White Cells.—Average number in cu. mm., 8–10 thousand; varieties (after staining):

1. *Lymphocytes*—about the size of red cells, as flat objects present narrow basophile rim, homogeneous or reticulated, about compact or reticulated spherical nucleus; granules absent but reticulum may show nodal thickenings; no nucleoli. Vary from 22 to 25% of total white cells.

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2. *Large mononuclear leucocytes*, may be largest cells in normal blood, protoplasm slightly basophile, finely reticular with nodal thickenings; nuclei vesicular, circular, horseshoe or elongated, coarsely reticulated. From 2–4 %.
3. *Polynuclear*, two or three times diameter of red cell, protoplasm reticulated with neutrophile granules, reticulum itself slightly basophile; nuclei elongated and constricted into two or more lobes with connecting threads of chromatin, coarsely reticulated with central nodal thickening. 70–72 %.
4. *Eosinophile*, vary in size, protoplasm contains granules which are strongly acidophile; nuclei bilobed usually and coarsely reticular. 2–4 %.

In pathological blood occur also:

5. *Myelocytes* with neutrophile or eosinophile granules, mononuclear, in three forms:
    - (*a*) Ehrlich's myelocytes, medium size, nucleus central and pale, neutrophile granules; in leukemia and secondary anemia.
    - (*b*) Cornil's myelocytes, larger than no. 3, nucleus eccentric and pale, granules neutrophile; in myelogenous leukemia.
    - (*c*) Eosinophile myelocytes, like no. 4 but nuclei single; in myelogenous leukemia (granules may be excessive in size), malaria, myxedema.
6. *Mast cells*, mono- or polynuclear, contain large and small strongly basophile granules, metachromatic with many stains (thionin); in myelogenous leukemia.

The behavior of these cells with various stains is important. Anilin stains may be divided as follows (Ehrlich):

1. Basic stains, which unite as bases with the acid principles of cells (nucleinic acid); in the order of selective power, hematoxylin, methylene blue, thionin, etc.

2. Acid stains, which unite with basic principles in cells, as eosin, fuchsin, aurantia.
3. Neutral stains, usually mixtures of the two previous kinds, which show affinity for certain parts of cells, as artificial indigo (from propiolic acid) and the "triacid" stain.[1]

Classified according to the reaction of their granules leucocytes may be divided into:

A. Basophile, including mast cells, lymphocytes and large mononuclear leucocytes.
B. Neutrophile, polynuclear leucocytes, myelocytes.
C. Acidophile, eosinophile leucocytes and myelocytes.

Variations in Number.—Excess of polynuclear cells is called *leucocytosis* with or without the word polynuclear prefixed; excess of lymphocytes, *lymphocytosis;* if more than one kind, *mixed leucocytosis;* of eosin cells, *eosinophilia.* Physiologically there is leucocytosis in the new-born, after ingestion of food and during pregnancy. Pathological leucocytosis occurs with fever and inflammation, after hemorrhage, in cachexias and just before death.

Blood Plates.—These are oval or circular in outline, colorless, homogeneous or granular, 1–3 μ in diameter, and stain with acid and basic dyes. They are cohesive, of relatively high sp. gr., and are supposed to aid in formation of fibrin (coagulation). The number is estimated at 180,000 to 500,000 per cu. millimeter, and they are supposed to increase in afebrile anemia, leukemia and hemorrhage, to decrease in fever, malaria and after various poisons. Their exact significance is unknown, but their origin (perhaps not in all cases) is from extrusion of chains of globular material by red cells; this material gives some of the reactions of nucleo-proteids.

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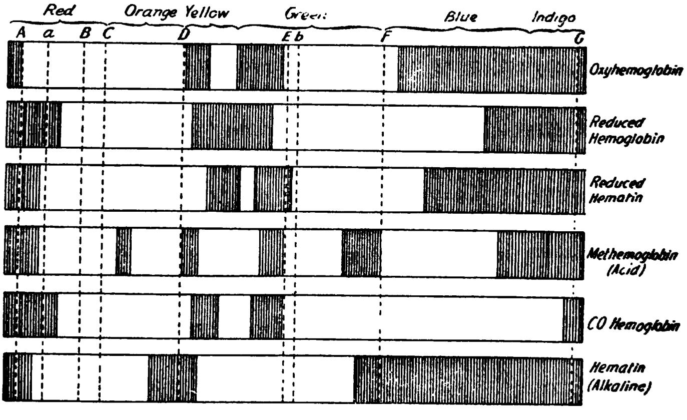
[1] Ehrlich considers the basic dyes as chromatin stains, the acid as plasma stains, and the neutral as given above; but the method of use and other considerations modify these views. For instance, orange and acid fuchsin are acid dyes, but by regressive staining they may color chromatin as well as plasma, safranin and nigrosin stain plasma though basic, when used with mordants. (Lee, M. V. M., p. 194.)

Hemoglobin.—The estimate of this body is of importance in anemias and certain poisonings. It may be accomplished in one per cent. solutions by the spectroscope, which shows both the blood pigment and also its particular form. Blood recently clotted may be dissolved in water; older clots by maceration in acetic acid, which gives the acid hematin spectrum; clots which have been exposed to heat should be macerated in ammonia, with the reduced or alkaline hematin spectrum resulting.

In fresh blood with the hemoglobin (Hb) unaltered, the spectrum of oxyhemoglobin is obtained, with an absorption band at *D*, rather thin, near the orange, and another at *E*, broader, near the green. The indigo and most of the blue are absorbed. With the addition of reducing agents (as ammonium sulphide) the color of the fluid darkens and the spectrum shows a broad band between *D* and *E*, less absorption at the blue end and more at the red—the spectrum of reduced hemoglobin. This test is positive indication of the presence of blood.

FIG. 5.



SPECTRA OF BLOOD PIGMENTS.

Hematin is produced by the action of acids and strong alkalies on reduced Hb. In acid solution its spectrum is similar to that of acid methemoglobin; in alkaline, it gives a broad band at *D*.

Blood containing methemoglobin may be chocolate color in the gross. Its spectrum in acid or neutral solution gives four absorption bands, one quite sharp between *C* and *D* in the orange, a fainter band in the yellow to the right of *D*, a broader band, more distinct, to the left of *E*, a fourth broad band to the left of *F* which may merge with complete absorption of the blue end. Carbonic oxide hemoglobin gives a rosy or cherry red color to both venous and arterial blood. In .5 per cent. dilution the spectrum resembles that of oxyhemoglobin but the bands are broader, the *D* band is displaced to the right, and there is less absorption at the blue end. On addition of ammonium sulphide to normal blood the spectrum of reduced Hb is obtained, that of CO-Hb remains unaltered. Corroborative tests should be used:

1. Warm the specimen with 10 per cent. solution of NaOH; normal blood becomes brownish green; CO blood turns cloudy, then clear red, with red flakes on the surface.

2. Zinc chloride solution colors normal blood black; CO blood bright red.

3. Dilute 4–5 times with lead acetate; normal blood becomes chocolate colored, CO blood red.

The estimation of the percentage of Hb in blood requires special apparatus and training. Miescher's modification of Fleischl's hemoglobinometer is as accurate as any and its technical difficulties trifling.

Special Pathology of the Blood.

Chlorosis is a disease which occurs chiefly in young women from defective hematogenesis, affecting principally the hemoglobin and secondarily the number of red cells. Arterial hypoplasia, auto-intoxication and functional nervous disorders have been supposed to favor it; heredity, poor hygienic surroundings and the establishment of menstruation are important; with the predisposition, an imperfect absorption of iron exists.

Blood Changes.—Total blood perhaps reduced (oligemia), sp. gr. diminished in proportion to the loss of Hb, low Hb index constant, in average cases Hb ranges between 35 and 45 per cent., index about .5; red cells slightly reduced, may be variations in size and shape, poikilocytes in severe cases and also nucleated red cells.

Progressive Pernicious Anemia.—This name includes all grave anemias in which the megaloblasts exceed the normoblasts or at least 33 per cent. of megalocytes are present (less in remissions). Its causation is undetermined (cryptogenic) in many cases; others coincide with chronic gastritis, gastric carcinoma, intestinal parasites (bothriocephalus latus, anchylostoma), repeated hemorrhages, and pernicious malaria, syphilis, prolonged diarrhea, typhoid and yellow fever. The essential lesion is metaplasia of the lymphoid marrow with production of megaloblasts; such metaplasia is invariably present, may resemble tumor formation, and is pathognomonic. The lesion in the blood is defective formation of cells and their destruction by some hemolytic agent.

Blood Changes.—Red cells from 1,000,000 to 200,000, megalocytes with increased Hb from 33 to 90 per cent.; megaloblasts very scarce, or numerous or outnumber normoblasts; microblasts rare. In fresh blood coagulation is feeble, rouleaux of red cells do not form, ameboid movement of poikilocytes may be present, of microcytes usual; degenerative alteration of red cells common. Leucocytes reduced, number may rise before death. Sp. gr. as low as 1.027.

Leukemia is regarded as a primary disease of the blood-producing organs principally affecting the marrow and the lymphoid structures. The essential process consists of hyperplasia of myelocytes or lymphocytes, or both varieties of cells, with secondary increase in leucocytes, secondary lesions in viscera, and leading eventually to disturbances of red cell production and pernicious anemia. In the myelogenous type the marrow and the neutrophile myelocytes are chiefly involved, in the lymphogenous type the lymph nodes, spleen and lymph cells of marrow are most affected and red cells suffer less early. The types may be mixed, as the disease affects one or the other group of structures at different stages. The apparent resemblance between leukemia and neoplastic formations, and cases of lymphemia arising in connection with lympho-sarcoma, are of great but still undeciphered interest. (See pp. 106, 111.)

Blood Changes.—But slight alteration in bulk, color usually pale, coagulates slowly (at autopsy), the red cells settling and leaving white cells and fibrin in a thick layer above them, with clear serum above all.

Red cells reduced to two to three millions or lower, deficient in Hb, size uniform, rouleaux normal, normoblasts fairly numerous, smear on slide peculiarly opaque and granular from excess of leucocytes. In late stages red cells show alteration in form with degenerative changes. Hb lower than normal.

White cells increased to 100,000–200,000 or more. Neutrophile forms occur as large mononuclear with central well-staining nucleus, abundantly present, and as large myelocytes with pale excentric nucleus; they may be lacking in neutrophile granules or show hydropic degeneration of the nucleus. Polynuclear neutrophiles occur from 17 to 72 per cent. (average 46 per cent.) and seem to vary inversely as the myelocytes; in the lymphatic form of leukemia they are very scarce. Degenerative changes in these cells are common; increased cohesiveness, paleness of nuclei, fragmentation of nuclei, and hydropic swelling. Eosinophiles are increased in the myelogenous form, varying from 3,000 to 100,000, hence of diagnostic value; ameboid movement well marked. In acute leukemia of both types, and in chronic lymphocythemia, they may be very scarce. Eosinophile myelocytes with granules of unequal size and staining affinity are pathognomonic of myelogenous form. Lymphocytes vary in numbers and proportion, but are less in proportion as the myelogenous character of the disease prevails. They vary from 2 per cent. to 10 per cent. Large mononuclear leucocytes occur but seem to lack significance. Degenerative changes are common in these cells. Mast-cells are more numerous than normally but may require search; they may exceed the eosin cells.

Special Characters of Blood in Lymphatic Leukemia.—The lymphocytes are the only form of white cells appearing in increased numbers and in some cases determine the nature of the disease without question; but the causes of chronic leucocytosis are so numerous that caution is necessary. They are usually of small size and normal structure and may attain 80–90%. In rare cases they may be large, especially in children; in chronic cases and adults the small prevail. Myelocytes, mast-cells and polynuclears may be very scarce, nucleated red cells are scanty.

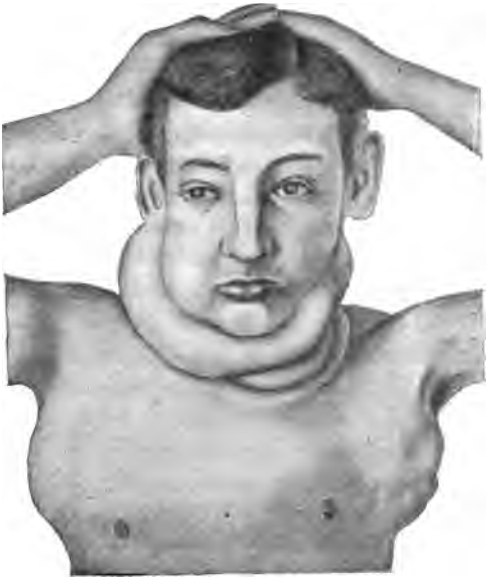
Both the numbers and the proportion of white cells may vary much in the course of the attack, owing to intercurrent disease. Spontaneous changes also occur and the disease may pass into pernicious anemia, just as pseudo-leukemia may pass into true leukemia.

The sp. gr. may be reduced with loss of Hb or restored by other albumins; alkalinity may be diminished, fibrin may be present in excess, peptone, nuclein, xanthin and uric acid have been found in the blood by various observers. Charcot-Leyden crystals are present in the blood during life and in all tissues (spleen) and exudates after death.

Pseudo-leukemia differs from leukemia in the absence of marked leucocytosis and in the hyperplasia of all lymph tissues, with metastatic growths in various organs. The anatomical lesions have included simple inflammatory hyperplasia of lymph nodes; lymphadenoma, and lympho-sarcoma. Until the etiology is definitely ascertained, the transitions between pseudo-leukemia, and hyperplasia of lymph nodes, leukemia, lympho-sarcoma and pernicious anemia, seem to indicate that they are closely related and perhaps have some causative factor in common.

Blood Changes.—Red cells little diminished at first, become less numerous as disease advances, but relatively not very pronounced anemia; cells may be undersized; nucleated forms uncommon. Hb always diminished. Leucocytes normal or diminished with a tendency to lymphocytosis; but there may be leucocytosis up to 50,000–60,000.

FIG. 6.



HODGKIN'S DISEASE. (*Coplin.*)
Cervical and axillary nodes enlarged.

Anemia Infantum, von Jaksch's Anemia.—This occurs in infancy and resembles leukemia without the visceral lesions.

The red cells are diminished, nucleated forms numerous, leucocytes generally increased. The spleen is enlarged with hyperplasia of its elements. The liver may be large and the lymph nodes also.

The Blood in Fevers.—The red cells are diminished in number, partly due to destructive effect of high temperature, coagulability diminished or increased, albumins diminished; hydremia and decreased alkalinity claimed by some observers and denied by others. The blood may contain the organisms of the disease in question.

Hemophilia is an inherited abnormality of the blood or the vessels or both which leads to severe spontaneous hemorrhages or great and uncontrollable bleeding after very slight wounds (extraction of teeth). The condition passes from males to males through females who are not affected. Thinness of the arterial coats and narrow caliber, disproportion between the total blood and the capacity of the vessels, local vascular hypoplasia, and obscure nervous conditions, have all been supposed of importance in etiology. The blood takes longer to clot than normally, but may not be deficient in fibrin. No constant alterations in the cells or the composition of the blood have been demonstrated.

Purpura Hemorrhagica, Morbus Maculosus, Werlhoff's Disease.—This is a disease of unknown etiology, though it appears as if certain cases were connected with the presence of bacteria, pyogenic chiefly. It is characterized by bleedings into and from skin, mucous and serous surfaces, high temperature in some, no temperature in others, and usually a rapidly fatal course. The blood clots slowly and imperfectly, leucocytosis is present in some cases, blood plates are at times scanty. Afebrile cases sometimes resemble pernicious anemia in their blood characters, but differ from it by absence of megaloblastic changes in blood and marrow and the clinical symptoms of marked hemorrhage. From the febrile form they differ in presenting less leucocytosis, more hemorrhage and no infection symptoms. The red cells are much reduced in number, small, nucleated forms rare; leucocytes normal or reduced, with large per cent. of lymphocytes.

Scorbutus; Scurvy.—Beyond the association with imperfect and improper diet the cause is unknown. Anemia, ulcerating gums and loss of teeth, bleeding in skin, mucous and serous membranes, general fatty degeneration of viscera, enlarged spleen, edema and hemorrhage in joints, may occur together or with certain features omitted or increased. The blood shows decrease of red cells corresponding to the hemorrhage, pale and broken cells and microcytes may be present; Hb is decreased; leucocytosis relative or absent.

Parasites.—The chief forms occurring in the blood are the bacterial, in septic diseases and relapsing fever, and in these only at short and uncertain intervals; the malarial organism which completes its human cycle in the fluid, the spirochete of relapsing fever, and the filaria sanguinis hominis, which occurs chiefly at night and after rest in bed. (See Vol. I., Chaps. VIII. and IX.)

CHAPTER II.

SPECIAL PATHOLOGY OF THE RESPIRATORY SYSTEM.

The Nose and Adjacent Cavities.

VARIOUS degrees of *malformation* are encountered in the nose, from complete absence with one-eyed infants (cyclops) to good external formation but lack of the septum or one or more turbinate bones, deviation of the septum and closure of one or both nostrils by bony growths, or by folds of mucous membrane. The extreme cases occur in non-viable infants; the less marked are amenable to surgical care.

Circulatory disorders are common in the nose, the rich supply of blood-vessels in parts of its cavity disposing the mucous membrane to sudden hyperemia and inflammation; more chronic congestion leads to various hypertrophies. In either form of congestion hemorrhage is common, giving all degrees of epistaxis. Such bleeding from the nose is observed in typhoid fever, purpura, hemophilia, pernicious anemia and diphtheria. Usually its source is a ruptured vein, caused by external violence, blows, efforts at clearing the passages, and if excessive and repeated may be the beginning of profound anemia or even fatal. The blood usually appears externally, or is partly caught in the mouth; if patients are weak or asleep the blood may be drawn into the larynx or aspirated into the lungs. More often it is swallowed.

Inflammation of the nasal mucosa occurs as an acute catarrh from various simple causes, direct and reflex, with a stage of active hyperemia and dryness, followed by free outpouring of altered discharge which may excoriate parts it bathes, as the lip and nostrils; this contains mucin and white cells with countless bacteria at first, but in later stages may become purulent and epithelia are found. Attacks of this nature frequently repeated are apt to end in chronic rhinitis and extend to the posterior pharyngeal wall. Streptococci and staphylococci, diplococcus pneumoniæ and other forms are almost constant in the healthy nose, and it is probable that when the conditions favor they may set up the inflammation just as the colon bacillus, usually a harmless inmate of the intestine, may cause severe suppuration when the normal resistance of the intestinal wall is lessened.

Nasal hyperemia and inflammation may be due to vaso-motor disturbance in the territory of the trigeminus, and such conditions show a marked annual periodicity in some persons, occurring regularly every summer as certain plants ripen and scatter their pollen; among these the ordinary ragweed (ambrosia) has very irritating pollen.

Chronic rhinitis may follow the acute form simply, but is often connected with depressed states of the system. It is characterized by stasis in the numerous veins, hypertrophy of the membrane and increase in the size of the glands; it is usually localized about the lower turbinated bones and adjacent septum but may be general. After a course of years the newly formed fibrous tissue shrinks and an atrophic stage begins with destruction of glands, retention cysts of their ducts, thickening of the vessels and decrease in their lumina. The most severe grade of such a process appears in *rhinitis atrophica fetida*, ozena, as commonly encountered in syphilitic and tubercular children; the altered and partly purulent secretion combining with the ulceration of the cartilages and turbinated bones produces an extremely disagreeable odor. The septum may be lost and the bridge of the nose obliterated, so that the nostrils look straight forward. The bacteria found may be the usual pyogenic or the pneumonic cocci, or saprophytes which cause the decomposition of the altered excretion.

Infectious Granulomata.—*Syphilis* usually causes a coryza in the earlier stages which differs but little from any acute rhinitis; later there may be superficial ulcers on the mucous membrane or perichondritis and periostitis of the bones about the nasal cavities, or gumma, which lead to necrosis of parts of the nasal wall and indolent ulcerations. Such ulcers are common on the septum and often perforate it. *Tuberculosis* is met with in several forms, as a general scrophulous rhinitis, or as localized polypoid growths and ulcers. Commonly it follows artificial inoculation by the use of handkerchiefs soiled with sputa, or from picking the nose, or lupus of the face may perforate into the nasal cavity, or the disease may appear there as a primary affection. The cervical lymph nodes are apt to become involved and caseous.

From the horse afflicted with glanders the bacillus mallei may pass to the human nasal membrane and cause nodules, "farcy buds," and ulcers to develop, and in both leprosy and rhinoscleroma the nose is apt to be involved sooner or later.

Tumors.—The simplest and commonest nasal tumors are polypoid growths composed of fibrous tissue and myxomatous material in varying amounts, and when the latter element is very fluid the resulting tumor is a cyst. Papillomatous growths develop in the lower third of the nasal channel as is the case also of fibromyxomata. Either of these may be combined with hyperplasia of the mucous membrane and glands, the latter usually widely dilated. Chondroma, osteoma, sarcoma and other forms occur, the latter usually starting from the wall of one of the adjoining cavities (as the antrum) and pushing its way out and down. Carcinoma is less common, but epithelioma may develop about the nostrils, as so often where skin and mucous membrane become continuous.

In the various cavities of the skull which open into the nasal passages, the frontal, sphenoid and maxillary sinuses, similar affections of the membrane lining them and of the bones occur as in the nose, but often their clinical importance is greater because of their distance from the surface and the limited space which confines the accumulating products of disease.

The Larynx.

Malformations of the larynx are not common or of great importance; the organ as a whole may be small in general aplasias connected with hypoplasia of the vascular system, cryptorchism, etc.; sundry parts may be lacking or unsymmetrical, as the epiglottis, or the sinus of Morgagni may be dilated as a large air-filled space (as in monkeys), or fistulous canals may communicate with the outside from imperfect closure of branchial clefts.

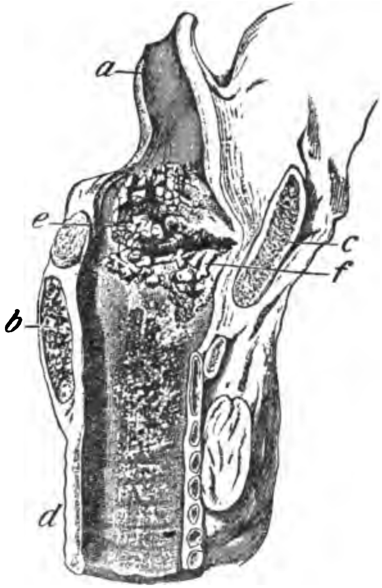
Disturbances of circulation are frequent. Anemia may be part of a general anemia, and often is the first sign of laryngeal tuberculosis. Active congestion of various grades follows inhalation of irritant gases and dust, the fatigue of long (and improper) use, and is the first stage of acute inflammation. Passive congestion occurs in circulatory disturbances from disease of the heart or lungs. Hemorrhages may be punctiform and limited to the submucous tissue, and are found after extreme acute hyperemia, from choking, in scorbutus and hemophilia; massive bleeding follows ulceration which opens a vessel of importance. Edema is clinically of great moment, especially in the young, because of the limited space and the importance of its function. It is common with passive hyperemia and chronic inflammation, but may develop suddenly with fatal consequences from irritants, inhalation of steam and gas, and in the course of nephritis and other disease accompanied by edema. The laryngeal picture is that of two pyriform masses corresponding to the arytenoïd cartilages and the aryepiglottic folds, which meet in the middle line and overhang the rima glottidis, closing off its posterior two-thirds at first and then the remainder; with each inspiratory effort these masses tend to be drawn downward and together shut off the supply of air completely.

Inflammation.—*Acute laryngitis* begins with hyperemia of the mucous membrane, increased secretion follows, the voice becomes husky or aphonic from imperfect vibration of the thickened vocal cords. (Strictly speaking these are neither cords nor bands, but triangular prisms, one side applied to the wall of the larynx, their upper horizontal surfaces visible in the laryngeal mirror, their sloping lower sides unseen, their thin edges approaching in the middle line and capable of tension and approximation by the muscular apparatus of the larynx ; being highly elastic they contract as the tension is lessened. They ought to be known as the vocal prisms, then, but long usage has sanctified the incorrect term "vocal cords.") The secretion is either increased mucus alone or contains leucocytes also ; if its proportion of pus-corpuscles is high the mucous membrane is usually very much swollen and shallow ulcers form on the posterior commissure and the posterior surface of the epiglottis. When the follicles are involved in such ulceration the resulting loss of substance is much deeper and healing is followed by scar formation.

Chronic laryngitis may develop from the acute process, often after repeated attacks, or slowly and independently. Slight varicosities are noticeable in the mucous membrane, the secretion is scanty and thick, the mucous membrane is thickened and from its swollen glands may have a coarsely granular appearance. On the vocal cords little patches of thickened mucus cling and the usual glistening look is lost ; small ulcers of mucous follicles are common. Such chronic laryngitis is often found in those who are obliged to talk long and loud (as auctioneers), in habitual drinkers and in those who suffer from pulmonary tuberculosis and other lung diseases. As elsewhere, an atrophic stage may follow the hypertrophic, and small papillary hypertrophies may occur and persist (verrucous laryngitis).

Croupous Laryngitis.—In exudative inflammation of the larynx, when the exudate contains fibrin and coagulates, with its contained pus-cells it makes a false membrane of grayish color and fairly tough consistence which clothes part or the whole of the interior of the larynx. It may be disposed merely as delicate patches or form a complete cast of the cavity and at first it is closely connected with the mucosa; such a membrane may be coughed out and reform in a comparatively short time. Microscopically there are fine and coarser filaments of fibrin, which stain with the Weigert method, entangling desquamated epithelium and pus-cells, seldom a few red cells also; under it the epithelium is lost and the glands are dilated from retained secretion.

FIG. 7.



PAPILLOMA OF LARYNX. (*Ziegler.*)

a, epiglottis; *b*, cricoid cartilage; *c*, hyoid bone; *d*, trachea; *e*, false vocal cord, *f*, true vocal cord, both bearing papillary outgrowths. Between them is the opening of the sinus of Morgagni.

Chondritis and *perichondritis* of the laryngeal cartilages is commonly a secondary affection after ulceration in the larynx, most often tubercular and syphilitic. At first but one cartilage is affected, then the inflammation spreads to others, the nutrition of the part suffers and the non-vascular cartilage dies. The pus and tissue detritus may exert a strong pressure on the larynx and interfere with breathing, or it may point and discharge through the larynx or externally.

Tumors.—Commonest of all tumors in the larynx are the papillomata, which resemble similar tumors of the external skin. They may be broad and but little raised above the surface or furnished with a pedicle and capable of rather free motion. They appear at any age but most often in adults, and are covered with flat or cylindrical epithelium or transitional forms. Mucous hyperplasia may result in the formation of polypoid tumors, on the edges of the cords or elsewhere, and the microscopic picture is that of fibroma. Mucous polyps differ in having enlarged and degenerated glands mixed with the fibrous tissue and, from the distention of these, the tumor may become cystic. True adenoma is not common, either here or in the nose. Malignant tumors may occur, though not very frequently as primary affections, and of these epithelioma is most common, starting from the vocal cords or from portions of the larynx which are covered with pavement epithelium. Metastasis to adjacent lymph nodes and the lungs is common.

Infectious Granulomata.—*Tuberculosis* of the larynx is usually an infection with the tubercle bacillus upon a chronic laryngitis; less often it is primary. It appears as a single ulcer, frequently about the posterior commissure or the posterior aspect of the epiglottis, while the rest of the larynx is free, or several ulcers in these and other sites, at times almost destroying the free edge of the vocal cords. If the ulcerative process extends to the submucous tissues fatal edema may occur, or perichondritis and necrosis of the cartilages. Between the ulcers the mucous membrane may be hypertrophic or even verrucous. A peculiar and unusual form of laryngeal tuberculosis occurs from extension of lupus.

Syphilis of the larynx, as in the nasal mucosa, varies from a simple catarrh to infiltration of both mucous and submucous tissues, deep ulceration and destruction of portions, as the epiglottis; or if the ulcers heal, firm scars of large extent may narrow and distort the part.

Leprosy occurs in the larynx as nodules or less often as a diffuse infiltration. Glanders produces small nodules which break down and leave deep ulcers; the epiglottis seems especially liable to such lesions but they occur deeper in the larynx also. Occasionally rhinoscleroma of the larynx has been encountered, in the later stages of the disease.

Foreign bodies may be solid objects, food, coins, buttons, etc., held in the mouth and accidentally inspired, or pieces of food misdirected in swallowing, or rags and other matters introduced (in the new-born) with criminal intent. Blood, vomit, pus, etc., may also be inspired and close the orifice temporarily or fatally.

Wounds.—The commonest are self-inflicted from attempted suicide and while not immediately fatal, even though the pharynx be opened, yet blood or food may enter and cause asphyxia or discharges may be inspired and set up a pneumonia. To this subcutaneous emphysema may be added. In hanging and choking the larynx may be injured from the squeezing, but usually it escapes by being lifted upward and backward into the oral cavity. In the aged, when the cartilages are calcified, breaks may occur more easily, and even the hyoid bone may be fractured; this is seldom the result of direct violence, more often it follows severe muscular exertion as in vomiting and is rare from any cause.

Trachea and Bronchi.

Malformation.—The only anomaly of development of importance is the occasional appearance of a fistula due to the imperfect closure of the third or fourth branchial cleft. The fistula opens by a small orifice on the anterior edge of the sterno-mastoid oftener on the right side, about 3–4 cm. above the inner end of the clavicle and may communicate with the trachea or end blindly in the tissues; the inner portion is at times much dilated, and is then termed a branchiogenic cyst.

The trachea is seldom diseased by itself; usually it is inflamed when either the larynx or the bronchi are inflamed and in a manner similar to the main disease. Tumors are not so common as in the larynx and infectious granulomata are secondary to similar lesions in the lungs or larynx. All that may be true, then, of the pathology of both the upper and the lower portions of the respiratory tubes will be true of the trachea and does not require special description.

Bronchi.

Circulatory Disorders.—The disorders of the larger bronchi present but little differences in their pathology from those of the trachea and larynx, but in the case of the smaller divisions of these tubes new factors enter which require special mention. The finer divisions are easily diminished in lumen when the lining cells are swollen, they are easily obstructed by inflammatory exudates, and inflammation in their walls almost always passes over to the tissue of the lung; hence capillary bronchitis should be considered with diseases of the lung. The larger bronchi are usually anemic on post-mortem examination, for their elastic fibers press the blood out; hyperemia may be acute and arterial from irritation by dust and other inspired matters, or very commonly it is passive and dependent upon cardiac disease, especially in lesions of the mitral valve. But a small portion of the blood from the bronchial wall returns by way of the venæ cava and azygos, the larger part returns by the pulmonary; hence any uncompensated obstacle in the left side of the heart must produce chronic passive hyperemia. This appears post mortem as a dark red or even brownish discoloration of the mucous membrane, swelling of its layers and increase of its secretion, the bronchi being filled usually with muco-pus; owing to the presence of air the color may be bright red and simulate active hyperemia. Hemorrhage may occur from the presence of foreign bodies, or as the result of ulceration of any variety; when an aortic aneurysm opens into a large bronchus sudden and fatal hemorrhage occurs, but this is strictly not a bronchial bleeding; if the patient survives the immediate effects a fatal result may occur from inspired blood and pneumonia. Similar loss of blood by way of the bronchi is relatively common in tubercular diseases of the lung and erosion of pulmonary vessels.

Inflammation.—*Acute bronchitis* may be primary or secondary to tracheitis. As in other mucous membranes, the first stage is an arterial hyperemia with swelling and lessened secretion; then follows a copious flow of thin mucus with but few cells or of thicker and tougher sputum with more pus and a yellower tinge. Such an inflammation at the two extremes of life is more serious than in vigorous adults; the danger increases inversely with the size of the tubes involved and when the smallest divisions are inflamed (capillary bronchitis, bronchiolitis) the mortality is practically that of pneumonia. Abundant sputa may be termed *bronchial blennorhea* when very thin and *pyorrhea* when thick and purulent.

Chronic bronchitis follows acute attacks or develops in connection with chronic diseases of the lungs in which there are persistent circulatory and nutritive disturbances, as in emphysema and some forms of tuberculosis. The mucous membrane presents the same appearances as elsewhere, swollen and congested layers, papillary projections (villous bronchitis) and at times also the atrophic condition of glands, mucosa and muscular elements. The secretion may be light and viscid or profuse and more purulent, and where the bronchi are dilated such secretion may collect for a long time and undergo decomposition.

Croupous Bronchitis.—This form of acute bronchitis may be due to the inspiration of irritants, but most commonly it follows diphtheritic infection; with every croupous pneumonia, and with laryngeal croup which descends, there may be a similar affection of the bronchi. The first stages may be like an ordinary catarrhal inflammation or with extreme dyspnea, and high fever; after the lapse of a few hours or days the peculiar sputa appear. The inflammation may affect the entire bronchial tree and in this case is of very grave prognosis. In the chronic form the disease may follow an acute attack of any bronchitis or it may develop in connection with chronic lung diseases and frequently it is limited to a certain section of the bronchial territory. Dyspnea is hence less marked in the chronic form. The characteristic sputum is of a yellowish-white color and when spread out in water reproduces the branching of the air passages; complete casts of large sections are not infrequent in the chronic form. Casts of the larger tubes may be hollow, those of the finer are solid, and sometimes made of concentric layers of coagulated material. This is often considered to be fibrin, but only the middle of a cylinder gives the fibrin reactions, while the rest is evidently thickened mucus. Tangled in the meshes of the coagulum are many white blood cells and degenerated epithelia and frequently the peculiar crystals which are thrown off in asthma, and found also in prostatic secretion, called Charcot-Leyden crystals. These are slender double pyramids (octahedra), at times bent on the edge or the flat or spirally, transparent, supposed to be related to nucleo-phosphoric acid and derived from leucocytes in most cases; they measure about 40 μ by 6–8 μ. Another element in the sputum is the Curschmann spiral, long twisted and very slender coagula surrounded by a fringe of very delicate fibers.

Diphtheritic bronchitis occurs as a complication of the same disease in the pharynx and larynx and the pseudo-membrane presents the same characters as elsewhere, or, more commonly, the process is croupous in the bronchi and diphtheritic in the pharynx.

Putrid or gangrenous bronchitis may accompany gangrene of the lung, due to infection by particles of necrotic lung tissue; the mucous membrane is softened and destroyed and hangs in decomposed shreds from the wall.

Peribronchitis and inflammation of the entire wall of the tube occur frequently with tuberculosis of the lung.

Stenosis and Dilatation.—The lumen of a bronchus may be narrowed by inflammatory and other swelling of the lining membrane and by pressure from without, tumors, cicatricial tissue, etc. The area of lung supplied may collapse (*atelectasis*) or become inflamed, or, if a little air can enter but escapes with difficulty, the lung tissue may be emphysematous. Foreign bodies also close the bronchial lumen more or less completely, with ulceration from pressure where they lodge, inflammation of the lung, perhaps gangrene; usually they lie in the right bronchus.

Bronchial dilatation (bronchiectasia) may be limited to but one or a few branches or distributed over large sections; the middle sized branches are oftenest involved and at the same time the finer divisions beyond may be obliterated. The dilatation may be sacculated, fusiform, cylindrical or irregular, and in the wall of the dilated portion nearly all the elements are atrophied; the mucous membrane may be swollen or thinned and its secretion is abundant and purulent or nearly absent. Fibrous thickenings about the dilated bronchus lend its walls an apparently normal size. When many of the bronchi are in this condition the lung tissue between presents degrees of compression, with irregular communicating passages through it, and this condition has been called *bronchiectatic phthisis*. Collections of mucus and pus may decompose and set up gangrene, or undergo drying and calcification. All forms of dilatation are usually associated with chronic bronchitis and lessened resistance in the bronchial walls, specially marked when the lung tissue is the seat of fibrous hyperplasia, for then the inspired air exerts a pressure upon the weakened wall of the tube and the fibrous tissue, contracting from outside, assists the dilating action.

Tumors.—In the bronchi primary neoplasms are very rare, while secondary are common from the ease with which the returning blood current may carry particles from distant tumors to the vessels of the lung. Carcinoma may develop from the muciparous glands of the bronchial mucous membrane, usually of a middle sized tube and in the lower lobes.

Infectious Granulomata.—Both tubercles and syphilitic scars are found in the bronchi, the former is an almost constant lesion in various forms of tuberculosis of the lung. A few or many shallow ulcers in the mucous membrane are found, some of which may pierce even to the cartilage or the outer layer.

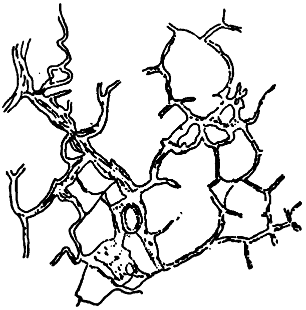
Lungs.

Atelectasis.—In consequence of debility the new-born infant may be unable to draw air into the lung and it remains in the fetal condition; injuries to the brain and cord during parturition and foreign bodies, as mucus and amniotic fluid, in the upper air passages produce the same result; or part of one or both lungs may be atelectatic and appear solid, small by comparison, bluish or brownish red, not crepitating, non-elastic. If the atelectasis is recent the portion involved may be blown up by forcing air in and resemble normal lung. In the adult, parts which have become atelectatic may be solid and tough, pigmented or not, compressed by fluid, adherent by the pleura to the side of the thorax or presenting a bronchus whose lumen is obliterated at some point. The bronchi show hypertrophied mucous membrane with villi and swollen glands and the cartilages may be relatively or actually hyperplastic. Any lung or part of a lung deprived of its air will sink when thrown into water. The fate of a non-aërated part is either inflammation or fibrosis and calcification.

Emphysema.—When an air passage is opened by a wound and the air from it gains entrance to the interlobular tissue of the lung, sometimes also after ulcerations, the condition is called *interstitial emphysema* and resembles a similar state of the subcutaneous cellular tissue. The lung appears pale and distended and under the pleura are small collections of air which may be pushed about by gentle pressure. Rapid decomposition produces the same appearance at times, the gas collecting under the pleura.

Vesicular or *substantial emphysema* is the name given to permanent dilatation of the vesicles and is a chronic disease with important secondary results. The dilatation may be due to loss of resisting power in the lung tissue, closely associated with imperfectly developed elastic tissue, and to continued and violent expiratory efforts. An *inspiratory emphysema* has been described as occasioned by a partial vacuum in the chest when the smaller bronchi are closed by swelling of the mucous membrane and the expansion of the thorax is unable to fill the alveoli. *Compensatory* or *vicarious emphysema* occurs when one portion of the lung is functionally incompetent and the rest exposed to greater air pressure because of dyspnea; it is common in the unaffected lung when the other is the seat of lobar pneumonia, or when adhesions and congenital atelectasis lessen the volume of a lobe. This vicarious condition appears especially on the edges of the lungs and close inspection discovers widely dilated alveoli under the pleura; where many of these coalesce it is termed *bullous emphysema.* The portions of lung which make the largest excursion during the respiratory act are the most often dilated and

FIG. 8.



EMPHYSEMA OF THE LUNG; LOW POWER. DILATED VESICLES WITH RUPTURED AND NON-VASCULAR WALLS.

the condition is found in both acute and chronic lung affections.

Two elements enter into the explanation of the gravity of true or alveolar or vesicular emphysema. The first is that despite the apparently larger volume of the lung the actual breathing surface is much decreased, because the dilated alveolar walls become thinner and thinner and gradually disappear; and secondly, in proportion to the extent of the former change, the number of permeable capillaries, and the total blood content of the lung, diminish. Such lungs on autopsy fill the thorax and do not collapse when it is opened, the ribs and sternum are in the position of full inspiration and the diaphragm is as low as the seventh rib, the lungs cover the heart and look pale and dry; the anterior sharp margin is rounded like the posterior, the entire lung is yielding and non-elastic, and the dilated alveoli and groups of alveoli are clearly apparent on gross inspection. With this condition nearly all the elements of the lung structure are atrophic, even the large vessels and the walls of the bronchi; with the latter there are frequent dilatations. Commonly also a chronic bronchitis is evident from the thick muco-purulent secretion in the tubes. As a general result of the imperfect use of inspired oxygen and lessened blood content of the lung, the condition of the patient suffers severely; and to this are added the effects of the mechanical obstacle to the venous current, such as dilatation of the hepatic vein and its radicles, imperfect liver function, edema of the feet and legs, dusky hue of the surface generally, cardiac hypertrophy and dilatation (right side) and serous exudates in various body cavities.

A form of emphysema in the aged is due to simple atrophy and diminished nutrition in the lung and the organ is decreased in volume.

Circulatory Disorders.—The double circulation through the lung is important to remember in this connection, the capillaries derived from the pulmonary artery subserving the function of the organ, respiration, while its nutrition depends upon the bronchial artery which supplies the walls of these channels and also the visceral pleura; anastomoses between the two are unimportant in normal conditions.

Pulmonary anemia may be part of a general anemia or may follow pressure from pleural exudate, tumor, etc., and atrophy of the capillaries as in emphysema; in the adult, especially the dweller in cities, the color of the anemic lung is gray because of the foreign pigment; in the infant in may be pink.

Hyperemia.—A form of congestion of the lungs affecting dependent portions is common in the cadaver and is known as *hypostatic congestion;* this usually is found in the posterior, *i. e.*, inferior, portions of both lungs when the body lies on its back. It is quite possible that a lung which was actively hyperemic during life should show the condition only in dependent portions after death, so that hypostatic congestion is not always without clinical significance.

Acute hyperemia of the lung follows excessive muscular effort, especially if protracted, and may lead to edema of the organ; it is caused also by any irritant which may be inhaled, and collateral congestion may follow sudden cooling of the body surface and lessened blood content of one lung by compression. It is the first stage of inflammation of the organ, or pneumonia, and is specially marked in the lobar form; such a congestion differs from simple hyperemia in that the lung is already friable.

Passive congestion usually follows chronic disease of the heart, especially mitral lesions and myocardiac degenerations; it is also observed with chronic bronchial inflammation. In marked degrees of such chronic congestion the lung takes on a peculiar color and becomes firm in consistence; in the alveoli and the septa are found granules and masses of pigment, at times free, at times enclosed in leucocytes, which are the remains of red cells which have wandered out of the overfull capillaries. To this condition the name of *brown induration* is given, or, as the interalveolar septa are usually thickened, it has been considered as a low grade of productive inflammation and termed the *pneu-*

monia of heart disease. In the sputa of such a case leucocytes bearing pigment often occur.

Hemorrhage.—On autopsy the evidences of bleeding in the lung vary from small punctiform hemorrhages which require careful search, to filling and partial destruction of a large section of the organ (lung apoplexy). The blood lies in the tissue of the lung, between the alveoli as well as in them, when it comes from the organ itself; when the source of the bleeding is external, as from operation about the upper air passages, the bronchi contain most of the blood, but little may be found in the alveoli; smaller amounts inspired may reach the alveoli. The seat of such collections is often in the lower portions of the lung, one or both sides, scattered dark red masses appearing, corresponding to one or more lobules, the central portions deeper in color than the periphery. Hemorrhage may result from injury, as puncture by broken ribs, and also after cerebral apoplexy from vaso-motor spasm.

Infarcts may be one or many, usually sharply defined from the rest of the tissue, prominent and superficial, lying under the pleura and often along the edge of a lobe, of a deep red color and firm to the touch; they are compared to a cone or wedge in shape, the apex in the tissue and the base external, but many other and irregular forms are found. The alveoli are stuffed with red blood cells and the septa show the effects of pressure, hyaline and fatty degeneration. The infarct may arise from embolism or thrombosis of a pulmonary vessel or in the course of chronic venous stasis. About the infarct there is usually a zone of inflammation, and if the embolus contains microörganisms, or they gain entrance from the air, suppuration and gangrene of the part may follow; in other cases the blood is partly absorbed, partly converted into pigment and the injury healed by new fibrous tissue which contracts later.

An occasional result of large *emboli*, usually coming from the inferior vena cava, is sudden death; the lungs being found pale, distended by the severe dyspnea which accompanies the case, and behind the embolus, in the pulmonary artery and the right heart, an excess of venous blood.

Fat embolism is observed in the pulmonary capillaries after fracture of large bones and destruction of the marrow, also in severe injuries to adipose tissue, burns and bruises; the fat appears in droplets and larger masses, liquid but caught in the capillaries, or even in large branches of the pulmonary artery. If the total amount of fat interferes with respiration it may produce death from pulmonary edema; smaller amounts may be pushed forward and lost in the arterial circulation or reproduce the embolism elsewhere, as in the brain.

Edema.—When there is a sudden outflow of serum into the tissue of the lung and the alveoli it is known as *pulmonary edema* and it may be acute and immediately fatal, or more slowly developed and less marked. The lung is paler than usual, unless at the same time congested, it feels firm, parts of it even resembling hepatized lung, and on section a frothy fluid escapes in quantity; both lungs or a part of one may be involved. With inflammation of part of a lung the rest is apt to become edematous. Pulmonary edema occurs as the final stage of heart and kidney disease and occasionally in alcoholic and insane patients. The cause of edema of the lung is frequently a loss of power in the left ventricle while the right continues to functionate, the extreme degree of stasis attained leading to rapid transudation. On dropping a piece of such lung into boiling water for half a minute the albuminous serum coagulates and may then be demonstrated between the alveoli as well as in them.

Gangrene.—After a portion of the lung has undergone necrosis (as coagulation) the access of saprophytic germs may set up putrefactive changes exactly similar to those which occur in the air in any dead tissue. Aspiration of decomposed tissue and pus from abscesses, perforation from the esophagus, cancerous or otherwise, and foreign bodies in the bronchi may be the exciting cause of gangrene; emboli from distant parts also produce pulmonary gangrene and cancer, and rarely tuberculosis may terminate in it. Alcoholic habits seem to predispose to pulmonary gangrene. Two forms of the lesion are recognized, the *diffuse* and the *circumscribed.* The latter appears as spheroid or ovoid foci, often multiple, of a black or green color and foul odor, surrounded by a zone of edematous and congested or inflamed tissue; a line of demarcation forms, the decayed tissue softens and separates from the rest and may be expectorated or persist as a thick broth of broken-down putrefied tissue. Through such a cavity a bronchus or a vessel may still pass, though the first shows inflammatory reaction or necrosis of its walls and the latter is filled with a thrombus; if the vessel is opened severe bleeding may occur. Should the edematous and inflamed tissues about such a focus take part in the gangrene the process spreads and we have the diffuse form which is rapidly fatal. Such gangrene is rarely anything but the moist variety on account of the rich supply of blood in the organ. It may pass over from the lung to neighboring parts, break into the pleura or pericardium, or cause emphysema of the skin of the thorax after adhesion between the two layers of the pleura and perforation. If the patient survives long enough septicemia appears and there may be embolism of distant parts and secondary gangrene. Although the prognosis is grave, yet a gangrenous process may be healed after evacuation of the softened material and suppuration and granulation of the cavity. Putrid bronchitis is a regular accompaniment of gangrene of the lung and the characteristic sputa, red or even purple, thin and foul, with an offensively sweet flavor, are pathognomonic. In a glass the sputa settle in three layers, the top foamy, grayish or yellow, the middle transparent and thin, the lowest reddish-brown or purulent or mixed with particles of lung tissue; in this the microscope discovers triple phosphate crystals, needles of margaric acid, fat drops and granules, elastic fibers, pus cells and pigment. The organisms may be staphylococcus aureus and albus, filamentous and branched forms (streptothrix), and ciliated monads.

Pneumoconiosis.—Workers in dust of many kinds are liable to inspire finely divided particles of inorganic matter. If coal dust is the chief element the condition is called *anthracosis;* iron and steel particles cause *siderosis;* workers in stone inspire quartz and other dust (*chalicosis*); millers and bakers inhale flour (*farinosis*). For a time the lung is very tolerant of such foreign material, and it is laid away in the interlobular connective tissue and the bronchial lymph nodes without apparent damage to the function of the organ; but beside the mechanical irritation, to which the response is commonly a fibrosis of slow development, the foreign particles may not be aseptic, or they may make microscopic wounds of the mucous membrane, and in either case more violent inflammation may follow. Mild degrees of bronchitis are frequent and the foreign matter is in great part expectorated with the secretion; in the alveoli a desquamative process goes on and the epithelia may degenerate and also be coughed out; cells containing pigment are usual in such sputa. After a time a form of lobular pneumonia may develop, or the already weakened lung is infected with the bacilli of lobar pneumonia or tuberculosis. Milder degrees of such dust inhalation are the usual thing in post-mortem findings; on the pleural surface there may be multiple miliary fibromata, in the lymph nodes the tissue is uniformly black, and through the lung in all fibrous parts similar pigment is found. Occasionally the dust passes into the general circulation and is excreted in part by the liver and in part by the intestinal mucous membrane. The contrast between the lung of the city dweller and that of the person always residing in the country, is very marked; in the infant the lung is bright red or pink and entirely free from foreign pigment.

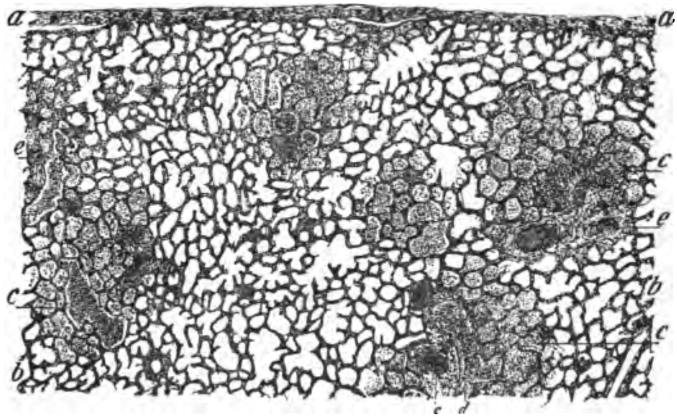
Wounds and Foreign Bodies.—When foreign bodies enter by the bronchi they are apt to set up an inflammation which involves the lung tissue secondarily, and this may end in gangrene. Entering the lung otherwise they imply a wound, and bullets, pieces of broken rib, or clothing, driven in by violence are probably commonest. Other wounds of the lung may result from severe compression and contusion of the thorax, stabs, etc. If of small extent and closed by pleural adhesions these may heal. Another variety of wound occurs from rupture of abscesses in the lung or adjacent viscera, as the liver, when the weakened lung tissue may be torn by respiratory and other muscular efforts.

Inflammation.—While *pneumonia* or *pneumonitis* is the name given to inflammation of the lungs, yet the disease occurs in so many different forms that it is necessary to classify them according to their anatomical or pathological characters: thus from the nature of the inflammatory product we have *fibrinous* or *croupous*, *catarrhal*, *fibrous*, *purulent*, *caseous*, and from the part of the lung involved we have *lobar*, *lobular*, *interstitial;* according to the path by which the irritant reaches the lung there are *broncho-*, *hemato-*, *pleuro-* and *lymphogenic* pneumonias, and when a visible excess of foreign material enters the bronchi it is called *inspiration pneumonia;* lastly, in the cachectic, blood settles toward the dependent parts of the lung and a *hypostatic pneumonia* arises. According to the clinical course of the disease we have acute and chronic forms, and from special bacteria tubercular and syphilitic inflammations proceed.

Lobular Pneumonia.—In children especially, bronchitis of the finer tubes passes over to the lung tissue and causes pneumonia; this is a common sequel to pertussis, measles, and other infectious diseases; in the adult it follows typhoid fever and is associated with some forms of tuberculosis; in old age it develops from chronic bronchitis; lastly, when matters of a septic nature are inspired they usually set up an inflammation of the same character, and it is possible for a patient with lobar pneumonia in one lung to inspire sputa into the other and thus present both lobar and lobular forms. The distribution of the disease in all these cases begins in single lobules and the air passages closely connected with them, the remaining lung tissue between being still supplied with air and capable of functionating. The affected lobules present in the alveoli serum, desquamated epithelia, red and white cells and mucus, the septa are infiltrated with round cells; the lobule is of a deep red color, prominent above the cut section, firm and friable, smooth on section, and non-aërated.

After a time the exudate undergoes granular and fatty degeneration and the part becomes yellowish or gray; normal tissue between may be blown up by way of the bronchi, the bronchi of the inflamed parts are closed by swelling of the mucous lining, or if still patent air does not reach the alveoli. The disease may begin in parts which have become atelectatic from closure of the bronchi. Developed from catarrhal pneumonia and often associated with it, one or both, we may have a purulent and a hemorrhagic form. The suppuration begins in the alveoli which are filled with exudate and is due to the entrance of pyogenic bacteria; the infiltration about the alveolus is more marked than in the simple form, the exudate becomes fluid and escapes or remains as an abscess. Large numbers of red cells in the exudate give rise to the other form; from either, gangrene may develop. If the pneumonia become chronic the alveolar

FIG. 9.



LOBULAR (ASPIRATION) PNEUMONIA. (*Ziegler.*)
a, pleura; *b*, normal lung; *c*, inflamed area; *d*, longitudinal section of a bronchiole; *e*, transverse section of an arteriole.

contents may undergo caseous alteration, due to the presence of tubercle bacilli, and there is then marked fibrous proliferation about the bronchi and alveoli affected. The influenza bacillus, anthrax and glanders frequently produce lobular pneumonia, and hypostatic and aspiration cases are usually of the same variety; experimentally section of the pneumogastric nerve causes the same form. No one bacterium is exclusively the causative agent; many kinds have been recovered from these lungs, cocci, and bacilli, but perhaps most commonly it is the streptococcus which is most active.

Lobar Pneumonia.—In this form an entire lobe or the major part of it is uniformly involved and there are no distinctions between alveoli which are filled with exudate and others which remain free scattered through the same part. In lobar pneumonia four well-marked stages may be distinguished—congestion, red hepatization and gray hepatization, followed by a stage of resolution.

Stage of Congestion.—The lung is intensely hyperemic, increased in size and firmness, abnormally friable, and contains little or no air; under the microscope the capillaries about the alveoli are filled to the utmost with blood, and within the alveoli there is forming a very delicate reticulum of fibrin, holding swollen epithelia and white cells in its spaces.

Stage of Red Hepatization.—The fibrin is more copious, the epithelia are wholly desquamated from the alveolar wall, and mingled with the exudate are numerous red and white cells. This exudate forms in each alveolus a tight plug, and as the elastic fibers are on the stretch, as soon as a cut is made in the lung these fibrinous plugs are forced a little way out of the alveoli and may be scraped off with the knife; the result is to give the section a peculiarly granular appearance. The part now contains no air and a small piece sinks at once when put into water; the total distention of all the alveoli makes the part as large as in full inspiration, and also very much firmer and heavier than in normal conditions. Other parts of the lung are the seat of congestion, edema and vicarious emphysema. This stage marks the height of the disease.

Stage of Gray Hepatization.—With the beginning of regressive changes in the exudate the color turns from red to gray or yellow. The red cells in the exudate have lost their color, the pressure from within the alveoli makes it impossible for the blood to enter the capillaries, although they are not obliterated, and the entire mass has begun granular and fatty degeneration; these changes gradually lead to the gray or yellow color which gives the stage its name, and as the inflammatory process advances it is possible to have all three stages present at the same time, the older parts being more nearly gray or yellowish-white in color. In alcoholic patients hemorrhages are frequent in the hepatized tissue, and here the red color persists. As degeneration proceeds the contents of the alveoli become fluid and are either expectorated or absorbed by the lymph-vessels. Of the three lymph-systems in the lungs, one opens by at least one branch into the lumen of each alveolus and there are also foramina between alveoli. Should a lymphangitis accompany the pneumonia, as is very common, and the vessels become stopped, resolution proceeds very slowly, since removal by absorption is greater than by expectoration. The complete disappearance of the exudate in favorable cases takes comparatively little time and the lung returns to a practically normal condition; but often the elastic tissue has suffered with the rest and the alveolus recovers its elasticity slowly and imperfectly. Croupous pneumonia may become purulent, either generally or in localized areas, but this is not common. An abscess formed in this way may break and discharge through the bronchi or into the pleura, causing empyema; or if single and small it may become encapsuled and calcified. In great constitutional depression, as cachexia, diabetes, senility, croupous pneumonia may end in gangrene. If the part of the lung involved lies directly under the pleura it also becomes the seat of a fibrinous inflammation; this may leave adhesions between the lobes. In the apices of the lung croupous pneumonia is apt to resolve imperfectly and here tuberculosis frequently is the sequel.

Croupous pneumonia is usually lobar, but it may occur as a lobular inflammation after influenza. The disease affects the right lung, and the lower lobes, in men, more than the contrary of these. Associated with such affections of the lungs there is usually congestion and swelling of the spleen, cloudy swelling or acute inflammation in the kidney, and at times meningitis, especially in children.

The bacterial form most often encountered in pneumonic lungs is the diplococcus pneumoniæ of Fränkel, in other cases the bacillus pneumoniæ of Friedländer, in others colon bacillus, pyogenic cocci, etc. Before these bacteria can cause an inflammation of the lung, there must be an irritant or some other factor present which reduces the local and general powers of resistance; inspiration of air at too low a temperature, vaso-motor reflex from the skin, and local or general disease are the commonest. The diplococcus pneumoniæ in other parts of the body may be pyogenic, as in meningitis, and as the exudate in the alveoli becomes more cellular after the fibrin coagulation is completed, the termination of the process as a suppuration is easily explained. A fatal termination of lobar pneumonia may be due to the extent of lung involved and loss of the respiratory function, and yet apparently complete double lobar pneumonia cases do at times recover; in other cases a toxic influence is more pronounced; or the heart may be overburdened, or the other lung may suddenly develop fatal edema; or gangrene and abscess formation end the case.

Interstitial Pneumonia.—The acute form may accompany either catarrhal or croupous processes, more often the former, and on the other hand a severe form may develop first and the alveolar exudate follow. This is not common but it may occur in septic conditions of the system, or follow suppurative processes in the neighborhood; as when a gastric ulcer perforates the diaphragm and attacks the adherent lung. Another variety follows septic embolism, but here the suppurative inflammation is confined to one main vessel and its distribution. If rapid in their course the first presents as fine and coarser lines of suppuration in a large meshed network all through the lung, the latter as a group of small abscesses or a single large one. The chronic form of insterstitial inflammation consists in the steady proliferation of new connective tissue along the course of bronchi and vessels and between the lobules and alveoli. Rarely it is the sequel to acute pneumonia of either form, or it results from the irritation of inspired dust, or appears as an essential element in the chronic changes of tuberculosis and syphilis. In the bands of new tissue pigment deposits are common, and, as they contract, the total pigment is contained in less space and the color of the lung deepens as its consistence becomes firmer.

Tumors.—Primary tumors are not common in the lungs. Of the benign varieties lipoma, fibroma and chondroma have been described. In leukemia there are often multiple miliary tumors in the lungs composed of small round cells in a reticular stroma; they resemble tubercle but do not become caseous and they are both whiter and softer. Sarcoma is rare; it may be of the small round-celled variety or spindle-celled, and probably has its origin in the peribronchial lymphatic tissue. Primary carcinoma may develop from the crypts of the mucous follicles in the bronchi, and epithelioma has been described beginning in the epithelium of the alveoli. Secondary malignant tumors are commoner, emboli from any organ reaching the lung and setting up similar tumors to the primary. Endothelioma may pass from the serous investment to the lung or begin in the lymph spaces of the organ itself. A form of carcinoma depends upon aspiration of fragments of a malignant tumor situated in the upper air passages; they may be multiple and the largest, and presumably the oldest, may be cystic.

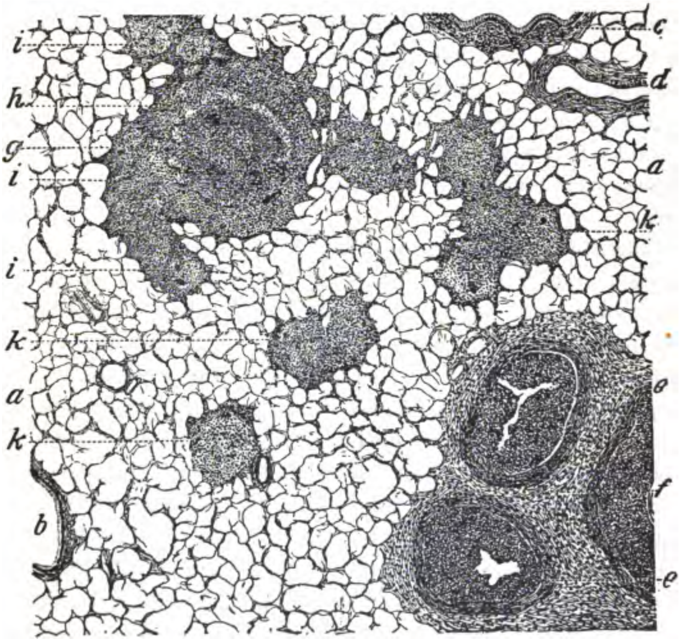
Infectious Granulomata. *Tuberculosis.*—Infection of the lungs by the tubercle bacillus may occur in three ways: through the bronchi, the blood-vessels or the lymph channels, termed *bronchogenic*, *hematogenic* and *lymphogenic* infection. However caused, the tuberculosis is a productive lesion, not an exudative. Tubercles may be found in any portion of the lung. In the connective-tissue elements of the organ tubercles resemble the same granuloma in any such tissue, made up of small lymphoid cells, larger epithelioid and occasional giant cells. In the alveolar parenchyma they present peculiarities, being irregular and elongated in outline, corresponding to the boundaries of the alveolus. The septa are thickened by round-cell infiltration, the alveolar lumen is reduced and its epithelial cells crowded together, leaving but a trace of the original space between ; at other times the epithelia multiply, show mitotic figures, and fill the lumen till the boundaries between tubercle and epithelial tissue disappear ; in such a mass giant cells often lie along what was the boundary of the alveolus. Lymphoid cells and pigment granules are also present. Such masses resemble pneumonic exudate which has become caseous, but the process is so clearly proliferative that the distinction is easy.

In the tubercle so established the usual degenerative changes occur—necrotic, hyaline and caseous—starting at the central portion, where nutrition is lowest, and spreading to the periphery ; fibroblasts surround the focus and invest it with a capsule. The vessels of the part are obliterated and not even permeable for injection masses.

Bacilli are found in such a tubercle both in the cells, giant and epithelial, and between them. Pyogenic cocci may not yet have reached the focus, and until they do there is no suppuration, no softening of the tubercle and ulceration of the lung tissue, and clinically but little disturbance in the general condition, emaciation, fever, or night sweats. In other words, many of the symptoms which are always associated with tuberculosis of the lungs are due to a mixed infection, the pyogenic bacteria attacking a tissue already weakened by the tubercle bacillus and rapidly destroying it. The tubercle bacillus is not cytotactic for polynuclear leucocytes.

Miliary Tuberculosis of the Lung.—As suggested above, the mode of entrance of the bacillus determines to a great extent the gross and microscopic characters of the disease. Tubercle granulations are most definite and typical when the infection enters by way of the blood current, hematogenic or disseminated miliary tuberculosis. The term miliary tubercle refers only to the size of the granuloma and the older yellow tubercles do not differ essentially from the recent gray translucent forms. A lung affected in this way presents small firm granules of a more or less transparent look scattered through lung tissue which is not apparently much affected, and the lesion may be totally or par-

FIG. 10.



PRIMARY TUBERCULAR FOCI IN THE LUNG. (*Ziegler.*)

a, normal lung; *b*, normal bronchus; *c*, bronchus with inflammatory infiltration; *d*, artery; *e*, caseous mass with fibrous capsule; *f*, fibrous hyperplasia; *g*, caseous center, and *h*, cellular periphery of a tubercle; *i*, *k*, tubercles invading lymphatics.

tially disseminated. If rapidly disseminated through the whole of both lungs it is usually only a part of a general tuberculosis, and other organs—spleen, kidneys, meninges and choroid—also present tubercles; in this case the tubercle may appeal to the sense of touch more than to sight, requiring a side illumination and careful search. More slowly developed chronic disseminated miliary tuberculosis may come from some focus elsewhere which allows a few bacilli to enter the venous current at intervals; as when a mesenteric or postperitoneal lymph node is attacked and bacilli make their way into the thoracic duct; here some of the tubercles are older and may show caseous changes. The lungs are still capable of functionating and between the tubercles may appear nearly normal; or lobar and lobular pneumonia may complicate the lesion, or simple hyperemia and edema.

When the entire lung is involved, as a rule the older tubercles, caseous and grouped, often surrounded by zones of caseous pneumonia, will be found in the upper lobes. Entering by the blood-vessels, the main distribution is along their course, and in the larger veins tubercles may be discovered in the wall on gross inspection; these may invade only the intima, and this fact makes the mode of entrance clear. The name embolic tuberculosis has been given to the condition without good cause. The source of such a bacillemia may be found in some of the abdominal or systemic veins or lymph nodes.

Localized dissemination depends upon invasion of a vessel in the lung itself, so that only the territory supplied by it contains the tubercles; thus a caseous bronchial gland or a tubercular process at the root of the lung may be the source of sudden dissemination through a lobe of one or the other lung. In other cases a tubercular mass as a center has distributed bacilli in a dwindling zone about it through the lymph channels, and such a distribution may occur as the result of invasion from a tubercular pleurisy; this process strongly resembles metastasis of malignant tumors.

When tubercle bacilli are inspired they are disseminated through the finer bronchi and their favorite place for settling is just where the bronchiole ends and the alveolar passages begin. Here the bacilli become attached and set up a bronchiolitis, or inflammation already present may offer favorable conditions for development; dust particles, especially with sharp edges, as fragments of iron, make microscopic wounds about which inflammatory reaction may start and thus favor infection. Probably phagocytes play an important part in all tubercular infections, carrying the non-motile bacilli into the tissues and setting them free to produce their specific lesions.

In the wall of the bronchus or alveolar passage so affected tubercles form which tend to spread in two directions, along the air passage, centrifugally, and into the lung tissue; the process becomes partly exudative as the alveoli about the bronchus become affected. Thus a series of little nodes, each with the lumen of bronchiole in the middle, is discovered on section, the centers caseous and a zone of pneumonia surrounding the whole; of course the true form is rather that of a ball or spheroid, presenting zones only on section. This distinguishing appearance of the lesion deserves the name of disseminated *peribronchitic tuberculosis* or *tubercular broncho-pneumonia.*

In the miliary form of tuberculosis the new-formed tissue is for a time protected from the entrance of pyogenic germs; in the bronchogenic form it is the regular and inevitable sequel to have a secondary infection with pus organisms, and the foci of disease soon begin to break down and soften under suppuration. The large amount of sputa reinfects other portions of the lung and the clinical course of this "stage of softening" is marked by great increase in the gravity of the symptoms. Such a course may not be followed in favorable circumstances, for a common experience in post-mortem examinations is to find small areas, chiefly in the apex of one or both lungs, where such a process has become latent, the tubercular focus surrounded by a thick capsule of new connective tissue

This is called "healed" tubercle and in many cases is practically so, but fresh infection from such a point is not uncommon. In other cases, where we may assume a single and limited infection, perhaps of attenuated bacilli, the course of the disease is very slow, though many tubercles be present throughout the lungs. Of all parts of the lung the apex is most frequently the seat of tubercular processes, partly because the ordinary and slovenly method of breathing does not fill this portion of the organ with each inspiration, and partly because the bronchi come off at such angles that they are easily obstructed by secretion.

Wherever the seat of the tubercular process, the regular development goes on slowly or quickly, with the softening and expectoration of the diseased tissue and the formation of cavities. The tubercle is non-vascular, owing to hyaline degeneration of the vessel walls of the part and the formation of thrombi, and hence degenerative changes are particularly liable to occur; some of the cavities however are bronchiectatic, the diseased bronchial wall yielding to the air pressure and the traction of fibrous tissue in proportion as the respiratory efforts are increased. Across the larger cavities, which contain semifluid and purulent material unless communicating freely with a bronchus, strands of firm tissue run which represent vessels, converted into fibrous cords by organized thrombi, and also fibrous bands of the stroma. Because of the obliteration of the larger vessels hemorrhage from the lungs is not so very common in the more chronic cases, occurring only when a vessel ulcerates before a thrombus has time to form and organize. The wall of the cavity is usually set with small tubercles and groups of them, and hanging from it are shreds of necrotic tissue; by destruction of intervening walls several cavities may unite to convert a whole lobe into communicating caverns, with strands and projections across them of fibrous nature. The fluid in such cavities may contain no bacilli, or none which may be stained; they require injection into a sensitive animal at times before they can be recovered. The size of the cavities and the number of them correspond fairly well with the clinical course of the disease, acute cases showing many small ones scattered all through one or both lungs. Such cases often develop from unresolved pneumonias which have become infected by many tubercle bacilli.

The converse of this is equally true, a lung which is already the seat of tubercle and cavity formation may secondarily become the seat of lobar pneumonia, which differs in itself not at all from lobar pneumonia in non-tuberculous lungs. Such a case should not be called tubercular pneumonia, but rather what it is, lobar pneumonia in a tuberculous lung. These cases are common in hospital patients in winter. It is not uncommon to have such a terminal pneumonia in portions of the lung which are still free, while the rest shows all varieties of tubercular infection, and, while commonly of the fibrinous form, it may be catarrhal.

The commonest complications of lung tuberculosis are those of general depression, anemia and emaciation, and secondary infections in various organs. The alimentary canal is in danger from sputa which are swallowed,[1] the larynx is affected in a similar way, the pleura shares in the tubercular process either acutely or chronically, the lymph nodes of the thorax seldom escape, and when a pulmonary vein is infected any organ of the body may receive and develop the bacilli thus entering the circulation. Intercurrent pneumonia and nephritis are common. Where the lung is infected secondarily to other organs the original seat of the process, as in the prostate gland, may remain quiescent, or the fatal termination of the case may be due to the primary rather than to the secondary disease, as in the intestinal forms.

Syphilis.—In the new-born with congenital syphilis the lungs may share in the condition with other parts, as the liver, spleen, pancreas, epiphyses, etc. In the adult the diagnosis is not always easy, for the case seldom comes to observation till it has existed for long periods.

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[1] Eighty per cent. of adult cases have intestinal lesions; about thirty-eight per cent. of children.

Congenital syphilis of the lung occurs as localized gumma, in a diffuse form, and in combinations of these. The diffuse may affect an entire lobe or the major part of it, giving it increased firmness and volume and a pale color. The microscope demonstrates a thickening of the stroma of the organ, both in the interlobular and alveolar septa, from the invasion of round and spindle cells; the vessels show uniformly a thickening of their walls, especially of the intima and media, and consequent decrease or destruction of their lumina; this explains the general anemia which has suggested the name "white pneumonia" for the process. By the pressure of the new-formed tissue the alveoli are crowded together and they may be filled with desquamated epithelia; the distribution of the lesion is often lobular and peribronchial. Circumscribed gummata do not differ materially from the preceding except in the spherical form of the focus and the greater tendency to caseous degeneration. Such gummata may be under the pleura or more centrally placed, most often in the lower lobes; in size they vary from one-half to several millimeters in diameter and, at first soft and white, tend to become firmer and more yellow with time; the center may be caseous or softer. A zone of inflammation may surround them.

In the adult the histological changes practically are the same. They show the same tendency to occupy the lower lobes and the right lung is more often the seat of the gumma than the left. The chief diagnostic points are that the gumma does not tend to liquefy under the attack of pyogenic organisms and that in it no tubercle bacilli can be found; while tubercular foci will, almost without exception, reward careful search with the presence of the bacillus, or it may be regained by inoculation of animals. Microscopically the gumma presents obliterating arteritis and phlebitis, and transitions from firm fibrous tissue to granulation tissue richly provided with round and spindle cells.

When such a gumma becomes secondarily infected with tubercle bacilli the diagnosis is especially difficult. Softening gummata may cause cavities to form and after the fluid material has been expectorated the cavity may contract and be obliterated by fibrous tissue; this distorts the lung, causing the pleural surface to dimple and changing the normal lobed appearance. The disease may be complicated by any form of bronchitis, pneumonia or tuberculosis.

Glanders.—The bacillus mallei may enter the lungs by way of the bronchi or by the vessels after systemic infection through the skin (the usual way); in the first case the inspired bacilli form small nodules in a lobular distribution, of a gray or pale yellow hue, soft and purulent if recent, at times caseous when older, each surrounded by cellular infiltration and punctate hemorrhages. The embolic or metastatic forms resemble the miliary abscesses of pyemia, occur in the lower lobes chiefly and are often surrounded by hemorrhagic pneumonia. In either case the entire lung may be affected or only a small section of it.

Actinomycosis.—Primary actinomycosis is less common than secondary. It may follow aspiration of the organism by itself or foreign bodies (as grain) infected with it. As a secondary disease it is due to rupture of an actinomyces abscess in the upper air passages or about the mouth, or the infection passes over from adjacent structures (mediastinum). In the latter case there is usually a single cavity, or a group of them, at the site of infection, containing thick purulent semifluid substance which contains small yellow points of fungus. Or, in the other modes of infection the distribution is more general and lobular, each nodule of indurated tissue is surrounded by pneumonia, often hemorrhagic, and tends to soften in the center; fibrous hyperplasia about these may make the resemblance to tubercle very striking.

Parasites.—The lung may contain fission fungi of various kinds as already mentioned, two forms of aspergillus (niger and fumigatus) give rise to a rare pneumomycosis aspergillina or exist as harmless inmates of tubercular cavities; mucor mucedo has also been discovered and various yeast forms in connection with carcinomata. Among the animal parasites cysticercus cellulosæ, strongylus and distoma hematobium occasionally occur; the latter causes a chronic hemoptysis in certain parts of the world (Japan, Korea, Egypt).

The Pleura.

Circulatory Disorders.—Independent changes in the vascular supply of the pleura can hardly occur, except perhaps in the parietal layer; these may be temporary and reflex from the thorax, on the principle that the vessels which supply a serous membrane supply also the integument over it. Practical application of this is made in the use of blisters and poultices to the chest. The visceral layer shares in anemia and congestion of the lung, stasis may lead to punctiform hemorrhages and the same occur in certain violent deaths, as hanging and choking; also in fevers and constitutional disorders with hemolysis. The cavity of the pleura may contain blood from wounds of either layer, bursting of aneurysms and perforation of tubercular and other ulcerative processes; this is called *hemo-* or *hematothorax.* The blood may be partially absorbed, partly coagulated and organized, or the bacteria mingled with it may set up various forms of inflammation.

Hydrothorax is the name given to the accumulation of serum in the cavity, not accompanied by inflammation. This often occurs as part of a general dropsical condition, and slight degrees are commonly associated with the end of nephritis and other chronic diseases. Acutely formed it may be due to sudden interference with either respiration or circulation, as in carbon monoxide poisoning. The two layers of the pleura are edematous, their lymph channels are dilated and the endothelial cells are swollen or desquamating; the lung may be compressed and even atelectatic if the amount of fluid is large and has been long present. The serum is clear and light yellow, or it may be slightly turbid from admixture of cells, and fibrinous coagula are found in it; the amount varies from a few c.c. to a liter or more (12 l. in a case recently seen).

Pneumothorax occurs when air enters the pleural cavity after perforation of either wall of the serous membrane, by tubercular processes, gangrene and abscess in the lung, rupture of emphysematous alveoli, perforation of the lung by purulent pleuritis, perforation by gastric ulcer, penetrating wounds of the chest. The tear may be round and with thinned or thickened edges, or linear; clear or partly covered with fibrin; large enough to be found readily by gross inspection or requiring a strong hand lens. The lung, unless held by adhesions, may be collapsed. When the cavity is opened the air rushes out with a hiss and if opened under water forms bubbles. The tension of the air pushes the other lung aside and the diaphragm downward, the intercostal spaces bulge and the entire half of the thorax is in expansion. When the condition persists, suppuration of the pleura, because of the bacteria present, converts the condition into *pyo-pneumothorax*.

Inflammation.—The pleura shares in the inflammation of the thoracic organs and walls, or is attacked in the course of general infections (acute articular rheumatism, typhoid), or becomes secondarily involved when the pericardium or the peritoneum is the seat of inflammation.

Pleurisy may be exudative, all proportions of serum, fibrin and pus occurring in the exudate. When fibrin predominates it is called *dry pleurisy* or *adhesive;* if the serum predominates it is called *pleurisy with effusion;* when the exudate is purulent it is named *empyema;* one and the same inflammation may pass through all these stages. Pleurisy begins with acute hyperemia, at times so marked as to cause petechial hemorrhages; the membrane soon loses its glistening appearance, becomes opaque and covered with a delicate film of fibrin, holding a few leucocytes in its meshes. This fibrinous coat may practically cover the entire surface of the lung and dip in between its lobes, fastening them together, or it remains in scattered patches. When the serous exudate is scant adhesions form between the two layers of the pleura, especially in the dome of the cavity. The serum is clear or blood-tinged. In some cases it accumulates rapidly till the thoracic contents suffer from the pressure. In the exudate there are always both red and white cells from the blood, but from the first, or after a time, the purulent element may exceed the others and the disease becomes empyema.

Beside the cellular elements present the exudate regularly contains urea, uric acid and cholesterin; in the diabetic, sugar also may be present. The exudate may remain for a long time in the cavity and at last be reabsorbed, or turn purulent; in the latter case the fibrinous layer on the pleura becomes infiltrated with pus cells and is called a *pyogenic membrane*, pus being continually added to the exudate from its internal surface. If the exudate is removed, by absorption or operation, the two layers of the pleura may become adherent over a greater or less extent, forming a dense white layer which at times obliterates the cavity; the vessels of the adhesions and false ligaments so formed are sometimes so numerous as to relieve circulatory disturbances in the lung.

Empyema, pyothorax, usually develops from the sero-fibrinous form of pleurisy, unless lighted up suddenly by entrance of pus into the cavity from abscess of the lung or thorax wall. The pus is yellow, grayish or green, acid in reaction, and later may be mixed with blood from thin-walled new vessels; it perforates and escapes through lung tissue and bronchi, into the abdomen, or externally; it may decompose either with or without communication with the air. Empyema accompanies carcinoma and gangrene of the lung, diabetes and pleural tuberculosis.

The results of pleurisy depend chiefly upon the amount and disposition of the exudate, also upon the variety. Dry pleurisy may be healed with adhesions of slight extent or larger and firmer ones; these restrict the movement of the lungs and if firm may contract the chest and lower the shoulder of that side. The adherent pleura may become extremely thick and its contracting force is marked and continuous; calcified plates may form in it. Between the lobes adhesions may be of little moment, or the starting point for tubercular infection. When serum is poured out in quantity it may be absorbed and the process ends as above, or it may become purulent and remain dormant, or cause symptoms of general intoxication; if it escapes by the bronchi, or externally, only adhesions and distortions remain, or a fistulous tract discharges indefinitely; in less favorable cases pulmonary abscesses and gangrene occur and end fatally.

Tumors.—Fibroma and lipoma may develop on the parietal layer, and chondroma and osteoma also; less often they occur in the visceral layer. Endothelioma may begin in the lymph channels and infiltrate a large section of the pleura, resembling in gross appearance the simple fibrous adhesions left by inflammation, or it occurs as small nodes and spheres scattered over a less thickened surface; the cavity may contain serum and fibrin. Metastases are found in the lymph nodes of the thorax, in the lung and liver. Secondary tumors are especially common with malignant disease of the mammary gland, passing to the pleura through the anterior chest wall or through the lymph vessels or as a series of interrupted nodules not connected with the primary tumor.

Infectious Granulomata.—*Tuberculosis* of the pleura follows similar affections of the lung or at times is a primary infection; even in many of these seemingly primary cases careful examination of the apex of the lung or of the bronchial glands will disclose the focus from which the infection came. The form is usually fibrinous, the hyperemic pleura with tubercles scattered over it being covered with a layer of fibrin and pus-cells in varying proportions; frequently there are small hemorrhages here and there. At times an ordinary pleurisy runs its course and afterward tubercles, lying till then latent under the pleura, become active and set up a tubercular inflammation.

Syphilitic pleurisy occurs almost without exception as an unimportant part of a general disease; it may take origin from subpleural gumma of the lungs.

CHAPTER III.

THE DUCTLESS GLANDS.

The Thyroid Gland.

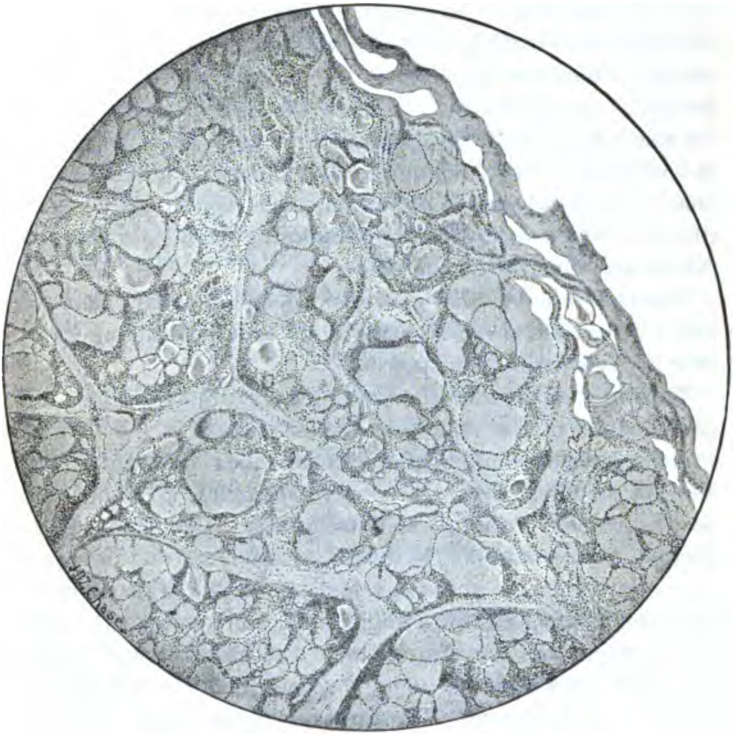
Malformations.—Entire absence of the gland has been noticed in some cases of cretinism; variations in its size and the number and disposition of its lobes are common, accessory thyroids occur near by or remote from the main gland, as behind the pharynx and lying on the arch of the aorta.

Circulatory Disorders.—*Active hyperemia* is favored by the free vascular supply and occurs especially in women, with swelling of the part; from repeated congestion the gland may enlarge (goitre), and this occurs alone or as a symptom in Graves disease. *Passive congestion* may be the result of heart disease.

Inflammation is not common. It may follow any exciting cause of inflammatory reaction and either resolve, form indurations or suppurate; the latter in the form of miliary abscesses sometimes accompanies mycotic endocarditis. If the suppuration is confined by the gland capsule it may become thick and calcified; if it breaks through it follows the plane of cervical fascia into the mediastinum, or perforates the trachea, esophagus or integument. From the swelling and pressure it may interfere with respiration before this and cause death.

Goitre.—The thyroid is peculiarly liable to hyperplasia, the resulting tumor being known as *struma benigna* or *goitre.* This may be the result of repeated active hyperemia, or the connective tissue may proliferate, together with great increase in the colloid material always found in the gland lumina. It is difficult to separate simple hypertrophy of the thyroid from simple adenoma and other enlargements of the organ. The name goitre is clinical rather than pathological and usually means simple hyperplasia. Struma benigna is divided into several secondary

FIG. 11.



SIMPLE GOITRE. (*Stengel.*)

Dilated acini, containing colloid material, separated by bands of fibrous stroma.

forms according to the gross appearance of the gland; it is called *nodosa* when special areas appear hyperplastic, *diffusa* when the entire gland is enlarged, *adenomatosa* when the gland acini are numerous and larger than usual, *fibrosa* when there is evident excess of new fibrous stroma, *cystica* when there are larger dilatations filled with colloid material; when colloid degeneration involves all the elements but the capsule a *colloid* variety is spoken of.

The results depend partly upon the size of the tumor but more upon the direction of its growth; those which develop toward the integument reach a large size without giving any great annoyance; the same degree of growth toward the trachea compresses its lumen to a slit; or when the thyroid passes in from the side between both esophagus and trachea it stops the passage of both food and air; if its main development is from the isthmus of the gland such compression effects appear early; other structures which suffer from the pressure are the jugular vein, the carotid artery and the vagus nerve.

Except for the fact that goitre is endemic in certain countries and is believed to have something to do with the drinking water, its etiology is entirely unknown.

Tumors.—Apart from the forms of hypertrophy, fibroma and combinations of the same with sarcoma occur in the thyroid. Primary sarcoma is often of the round-celled variety, but may also be spindle-celled; it is very malignant, invading adjacent tissues and forming rapid metastases. Carcinoma of the thyroid, *struma maligna,* is usually alveolar, starting in normal or hypertrophied gland tissue, rapidly invading the tissues of the neck and forming all into a single rather soft tumor. Degenerative changes are common in these cancers, fatty and colloid especially; less often the tumor becomes hard through the formation of fibrous tissue. Transitions from adenoma to carcinoma occur. Secondary tumors are uncommon.

Tubercles and syphilitic gummata have been found in the thyroid occasionally.

Simple degeneration, both colloid and calcareous, are at times described, and certain parasites have been found, as echinococcus and actinomyces.

The constitutional effects of thyroid disease are twofold, perhaps according to the absence or the excess of the gland's activity. In any case the relation of the gland to the nervous system is more evident than to the blood-making process, and it is permissible to think that it has a general control of important metabolic functions throughout the body. Thus when the thyroid is removed from young animals (rabbits) the bones cease to grow in length, and a similar observation has been made after operative removal in young children. Removal in the dog produces both nervous and nutritive disturbances, and in human myxedema the gland has been found markedly atrophied; it is known also, clinically, that artificial supply of thyroid gland lessens the symptoms of myxedema so long as it is kept up. On the contrary too much thyroid supplied will produce symptoms which strongly resemble exophthalmic goitre or Basedow's disease. In this there is evident hyperthyroidism, or overactivity of the gland, the rapid pulse, and bulging eyes and nervous symptoms being regarded as secondary to abnormal gland action; this disease has been considered as of strictly nervous origin, but the weight of evidence is against this view. In patients who have lost most or all of the thyroid a peculiar cachexia develops, called *cachexia strumipriva*, characterized by pallor of the surface, loss of hair, myxedema, weakness, and intellectual debility.

Cretinism is the name given to the condition of some children (especially in Switzerland) who suffer from thyroid disease (either atrophy or hypertrophy), imperfect growth and development, mental incapacity, swollen tongue and lips; the head is usually disproportionately large. The disease may be present at birth but more commonly it develops during the first year or so, and more than one member of the family may be affected. Heredity is marked at times.

Myxedema is a condition of adult life, developing without apparent cause, with or without evident change in the thyroid, taking its name from the chief symptom, a swollen and firm state of the skin. All the layers seem to be infiltrated with some gelatinous substance; though the impression is that of edema it does not pit on pressure nor does it affect dependent portions especially. The swelling of the face and tongue, the change in the voice, the loss of mental power and the falling out of the hair combine to alter the looks of a patient in comparatively a short time. The relation to the thyroid is evident from the effects of transplanting sheep's thyroid to the peritoneal cavity and administering the gland by the mouth. The hypotheses offered to explain the connection between thyroid and general disease vary between a possible toxic influence from misdirected gland activity, an excess of a normal product elaborated in the gland and delivered by the veins or lymphatics of the organ, which functionate as its ducts, and a gastro-intestinal intoxication from impaired control over the metabolism of mucin. (See Pt. I., p. 57.)

The Spleen.

Malformations.—Total absence of the spleen may occur in non-viable infants; the organ may be imperfectly divided by incisions along its sides; supernumerary spleens of small size are common.

Displacements.—Congenital or acquired conditions may be the cause of great displacement of the spleen. In complete situs inversus it may be found on the right side; hernia of the organ occurs through the anterior abdominal wall, or into the pleural cavity; increased volume of neighboring organs pushes it to one side or the other, fluid in the thorax and other causes of depressed diaphragm push the organ downward, food and gas in the stomach push it up and back; its own weight may drag on its supporting ligaments so that it comes to lie in the pelvis on either side. A normal organ will not depart so far from its place, but after chronic congestion, as in malaria, some degree of dislocation is the rule. The vessels stretch and become dilated and tortuous and if twisting occurs the organ may be atrophied or necrosed in its new location. It may also drag the fundus of the stomach out of place and produce compression of various parts of the intestinal canal.

Circulatory Disorders.—*Anemia* of the spleen is part of a general condition or follows great loss of blood; the organ is small and the capsule wrinkled, it is firm on section and no follicles may be visible, the stroma is somewhat prominent and the color is gray mixed with red. Recent embolism of certain vessels may produce a local anemia.

Hyperemia of the organ is physiological after the ingestion of food. Pathological hyperemia may occur in any acute disease of an infectious nature. The spleen is then swollen to several times its normal size, soft even to semifluid consistence, the stroma hidden in the swollen pulp, the capsule tense, even ruptured or breaking on gentle handling. Such increase in size and blood content is found in typhoid fever, anthrax infection, malaria, scarlet fever and diphtheria and some others of the acute fevers. According to the time in which it is developed this swelling is wholly hyperemic and edematous or fibrosis coexists; the density of the organ corresponds, being firmer, often with marked thickening of the capsule, when slow fibrous changes have acted with hyperemia. The excess of blood may gradually subside and the lymphoid follicles increase in size and number, the color of the section is then gray or brown or red with lighter colored granules scattered through it and lines of increased stroma. Microscopically the spleen of infectious diseases presents many large and small mononuclear cells, polynuclear leucocytes mixed with these and countless red cells; late in the disease many of the cells are swollen and cloudy. The hyperplastic organ shows many mitotic figures in the pulp and large mononuclear phagocytes with red cells englobed in them. The characteristic bacteria may often be recovered from the spleen, where they are in great part destroyed, either by phagocytic action or by antitoxic and antibacterial fluids. As a rule the swelling of the spleen gradually disappears after the disease has run its course, but it may not return to quite the normal size, the capsule may be permanently thickened and wrinkled, and the organ usually contains an excess of pigment.

Passive hyperemia and *stasis* often depend upon liver conditions which prevent the complete and rapid emptying of the splenic vein; to a slighter degree emphysema and valvular heart disease produce the same result. The corresponding changes in the organ are increase in size and consistence, thickening of the stroma and vessel walls throughout, and, upon contraction of the fibrous tissue, distortion of the organ.

Hemorrhage may occur in the organ itself after injury or as red infarction; infectious disease with rapid increase in size of the spleen may be accompanied by small bleedings. Loss of blood from the organ follows traumatic rupture of a swollen organ, as seen in the large spleen of malaria, after comparatively gentle blows upon the tissues over it, sudden extreme flexion of the trunk and compression of the spleen. A spontaneous rupture may occur with sudden hyperemia, especially when thickenings in the capsule prevent the expansion of the organ, as in old people; the usual result is the effusion of large quantities of blood into the peritoneum and death.

Infarcts may be red or white, septic or not, and are very common in the spleen because the anastomosis between the ends of the splenic arterial branches are so few; the veins are large and anastomose freely, hence the red infarct is more common than the white. If infected, the wedge-shaped area, which always has its base under the capsule and its apex toward the hilum, rapidly breaks down into an abscess and this may rupture into the peritoneum and set up general or local inflammation. The size of the infarct varies with the vessel in which the embolus lodges; it may be relatively very large, and there may be more than one. Simple infarcts may be absorbed in part and encapsuled by fibrous tissue, leaving only a somewhat stellate scar to mark the site; when the red infarct heals the blood pigment passes through various stages of decolorization and even in the scar leaves some trace of pigment.

Thrombosis of the splenic vein may follow similar process in the larger branches of the portal vein; it results in marked stasis in the organ. The splenic artery is often the seat of atheromatous changes and the nutrition of the part may suffer in consequence.

Inflammation.—*Splenitis* may be acute or chronic, localized or diffuse. The diffuse form is practically but a severe grade of the infectious hyperemic swelling which occurs in infectious disease. The organ may reach five times its usual weight, it is soft, even semifluid, dark red on section at first and later of a paler color; degenerative changes may go on in the pulp while fibrous hyperplasia proceeds in the stroma; the relative proportion of these determines the soft or denser consistence. Usually complete resolution is the termination of the process, but fibrosis, with permanent increase in size, or abscess formation and rupture, may be the results. In the form of circumscribed abscesses an "idiopathic splenitis" may arise after pus organisms enter the blood current; the resistance of the organ may already have been reduced by general disease and its toxin (typhoid). Septic emboli also produce multiple small abscesses in the spleen, as in malignant endocarditis, pyemia and severe typhoid. In size these abscesses may be punctate or several centimeters in diameter.

Chronic splenitis is almost always diffuse, produced in the course of chronic venous congestion from cirrhosis of the liver or valvular cardiac disease; it may also follow the acute form after infectious disease, and of these malaria is most commonly observed. Early in malarial disease the spleen is soft, dark red and swollen; later it shows diffuse hyperplasia of the stroma, thickening of the capsule, and pigmentation of a deep brownish tinge; the microscopic appearances correspond. The final stage of such fibrosis is contraction, as elsewhere, and the spleen is then very firm, pigmented, capsule thickened in patches and much wrinkled, showing that it has been larger; on section the bands of fibrous tissue make a network through the organ, the veins are narrowed and the lymphoid elements markedly atrophied.

Leukemic Hyperplasia.—In the *splenic* form of *leukemia* the organ undergoes a marked hyperplasia, probably starting in the usual way as hyperemic swelling, and in time the volume of the spleen may be fifty or more times the normal, the weight as much as 8–10 kilos. The first stage of the leukemic spleen is fairly soft and on the cut surface there is a seeming division into regions corresponding to the end distribution of arterial branches, slightly bulging and marked off by lines of stroma. Microscopically the blood-vessels are dilated and red cells occur in the pulp. Malpighian bodies and fibrous elements are hyperplastic. In the second stage there is evidence of granular degeneration in the pulp, and pigment granules gather about the Malpighian corpuscles; here and there anemic necrosis follows thrombus formation in the vessels. The gross appearance is a hardened organ with a much mottled surface, the capsule thickened irregularly, perhaps adherent to adjacent tissues, and the heavy organ is displaced, usually downward.

In *pseudo-leukemia*, *Hodgkin's disease*, the gross and microscopic findings in the spleen are almost identical with those of leukemia; from the organ alone it is hardly possible to make a diagnosis. Clinically there is more rapid formation of the lymph node hyperplasia and the blood examination is characteristic.

Operative removal of the spleen in dogs is followed by emotional disturbances, especially chronic anger. In the human subject splenectomy is remarkably well tolerated, perhaps because its blood-making function is performed by the marrow.

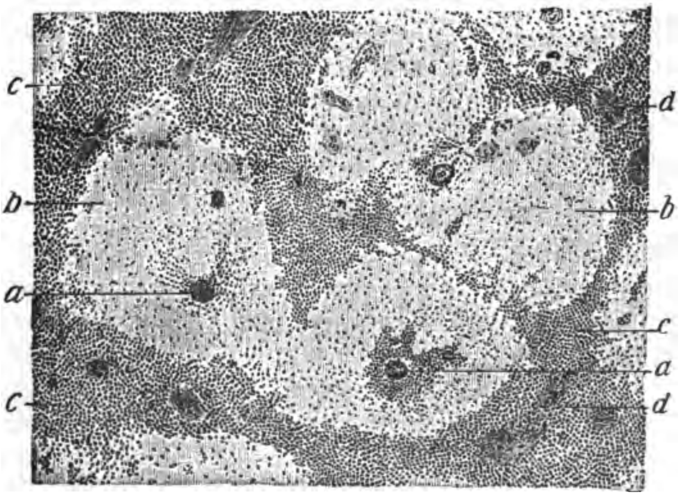
Tumors.—Primary neoplasms of the spleen are uncommon. Fibroma and angioma have been found. Among secondary tumors of malignant character sarcoma is least unusual, often melano-sarcoma; carcinoma may occur with general carcinomatosis and by extension from near-by tumors.

Infectious Granulomata.—*Tubercles* develop in the spleen as fine translucent points which resemble hypertrophied Malpighian bodies, but differ from them in their gray color, their abundance and the sharper definition from the pulp. In some cases there are large cheesy compound tubercles, as in chronic forms of the disease in children.

Syphilis produces increase in the size of the spleen, hardness of its tissue, and occasional gummata, miliary and numerous or single and larger.

Degeneration.—In the early years of senility the spleen begins to *atrophy* and this goes on more rapidly than in other parts of the body; the capsule becomes very thick, sometimes in patches, the lymphoid elements disappear, the fibrous are relatively more prominent and hence the organ is firm and small, its color brownish-red, and it is dry and pigmented.

FIG. 12.



AMYLOID DEGENERATION OF THE SPLEEN. (*Ziegler.*)
a, transverse sections of the splenic arteries; *b*, amyloid areas; *c*, spleen pulp; *d*, trabeculæ.

Amyloid degeneration is often most marked in the spleen or even limited to this organ. If the Malpighian bodies are specially affected they stand out above the cut surface as grayish translucent granules, resembling boiled sago grains and hence the name "sago" spleen. If the whole organ is involved it reaches a large size, its edges are rounded, consistence firm; the cut surface is anemic, dry, both firm and friable, and in color a peculiar red like that of fresh ham. By the iodine test the lymphoid elements become more prominent or the whole cut surface takes on the reddish tinge. Microscopically the degeneration begins in the walls of the capillaries and small arteries, converting them into structureless amyloid tubes, and between the thickened lines of stroma lie clumps of amyloid matter; few cells remain.

Pigmentation occurs in anthracosis when the foreign dust passes the bronchial nodes, by ulceration into a vein for instance; but the most common form is the deposit of malarial pigment, by which the organ may become dark brown to black on the cut surface. It results in part from destruction of red cells in the circulation and in part from their degeneration within the spleen.

Calcification occurs in the thickened capsule in old infarcts and in caseous foci.

Among parasites pentastomum denticulatum,[1] echinococcus and actinomyces are the commonest; all are rare.

The Thymus Gland.

Malformation.—In some cases the gland is extremely small or even absent, the latter with marked defect of other parts; or it may be large enough to reach deep into the mediastinum over the pericardium, or its division into lobules may be specially clear. Normally it begins to atrophy by the second year by a process of replacement of fat tissue, though traces of the gland may persist till the twenty-fifth year.

Hyperemia may be found in the new-born and punctate hemorrhages when death has been due to asphyxia. *Inflammation* is very rare and is usually limited to a localized abscess; these may be multiple after infection of the umbilical stump. In the *status lymphaticus* the gland shares with other lymph structures in the hyperplasia (see p. 114).

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[1] Larva of P. tenioides.

Tumors are occasionally found and among the malignant are sarcoma, angio- and lymphosarcoma.

Tubercles may invade the gland in general infection and as in cases of congenital *syphilis* caseous foci may occur.

The Lymph Nodes.

Circulatory Disorders.—Normally these structures appear pale in color though their supply of blood is fairly copious; with *anemia* they are clearly much softer than usual and on section dry. When *hyperemic* they are swollen; the color is reddish and the section very moist; the hyperemia may be limited to the outer portion or the whole cut surface may bulge from increase in its contained fluids. Extravasated blood in the nodes of a part after injury of various kinds is common. *Pigmentation* of the nodes occurs locally after the extravasation of blood in the collection territory, after the introduction of foreign pigment as in tattooing, and in some diseases of the skin associated with anomalies of pigment formation as in leucoderma syphilitica. Leucocytes are the chief carriers of the pigment but other cells with phagocytic powers assist. In the node the pigment granules lie in the lymphoid cells, in the stroma, or uniformly diffused; the result in marked degrees is impermeability for the lymph stream, so that it is directed to collateral channels and thus many nodes become tinged one after another, and also, if the foreign matter is irritating, the stroma of the nodes undergoes hyperplasia; if septic, ulceration follows.

Inflammation.—*Acute lymphadenitis* occurs most often from transport of microörganisms from peripheral traumatism, and the lesion is called primary when there is no inflammatory reaction at the seat of injury, mostly in superficial skin wounds, or secondary when it follows distal tissue inflammation. The first stage in the node is marked swelling and subjective tenderness, and as many of the prominent groups of nodes lie in the flexures of joints, large movements of the limb become painful. The swelling and hyperemia of the node give it a uniformly red and tumid look on section, and small points of hemorrhage may be found. Under the microscope all the spaces of the reticulum are stuffed with red cells and leucocytes, and while many of the latter are carried there from outside, others may be formed in situ, for mitotic figures are frequent in the round cells.

In favorable cases the evidences of inflammation disappear as quickly as they had developed; in others hyaline or fatty degeneration occurs; when there are frequent accessions of irritating material the gland may become indurated from fibrous hyperplasia. The presence of pyogenic organisms determines suppuration and in the nodes an abscess forms, with suppuration in the perinodal tissues and perforation to the exterior. In the superficial nodes the pus does no further damage, the cavity granulates and heals, but in deep laid portions of the lymph system severe consequences may follow perforation into one of the great serous cavities, into the air passages or the circulation. Such a swollen lymph node is commonly known as a *bubo*, and while most frequent after distal lesions, as with chancre, erysipelas and skin wounds, buboes may occur with systemic infection as in bubonic plague. Scarlet fever and diphtheria with an added streptococcus infection are often accompanied by inflammation of cervical lymph ganglia, and anthrax may cause severe hemorrhagic inflammation or even gangrene in the nodes corresponding to the site of infection. (See Lymphangitis, p. 47.)

The relation of many of the lymph ganglia to irritant and infectious substances may be regarded as a defense to the general system, for they filter out such sources of danger from the lymph stream and destroy them in situ, perhaps at the expense of suppuration.

Chronic lymphadenitis may follow frequent attacks of more acute type from recurring irritation and the nodes then are somewhat swollen, harder if the chief element is fibrous, softer if cellular increase coexists. Microscopically there are increase in the stroma with degeneration of the cells in the one case, and many round and epithelioid with some giant cells in the other.

Tumors. *Lymphoma, Lymphadenoma.*—While chronic hyperplasia follows chronic irritation, a more rapid increase in the elements and size of the lymph nodes, associated with similar changes in the spleen and variations in the composition of the blood, is observed in leukemia; the lymphatic form of that disease is characterized by great increase in the cells of the lymph ganglia generally, with a less pronounced hyperplasia of their stroma and vessels. The gross appearance of the nodes will vary with the proportion of stroma and cells, but they are always larger than normal and at times the capsule is broken through and the neighboring tissues share in the hyperplasia. In the late stages of the disease not only all the lymph ganglia of the body are affected, but in places where normally there is no lymphoid tissue, as in the kidney and the liver, metastatic formations arise which suggest an analogy with malignant neoplasms. The usual place for the lesion to develop is in a group of the cervical nodes and from here it spreads to adjacent and then to distant groups.

When the blood fails to present the characteristic lesions of leukemia and only the lymph tumors develop, perhaps with splenic changes, it is known as *pseudo-leukemia* or *Hodgkin's disease.* The new tissue resembles sarcoma (round-celled) and develops in situations normally free from lymphoid structures; hence the name malignant lymphoma or lymphosarcoma of some observers.

The two forms noticed in leukemia are found here also, a soft tumor whose nature is chiefly cellular increase and degeneration, and a hard form with marked fibrous increase. In the former the ganglia appear swollen but distinct from one another, soft and even fluctuating, furnishing a milky juice on section, at times presenting small hemorrhages; in the other the nodes are not so large and much firmer, the cut surface is fibrous and of grayish or yellowish color. It is said that these pseudo-leukemic tumors differ from the leukemic in their larger size, in their tendency to involve adjacent structures, in the larger size of their cells with more frequent appearance of giant cells, and in the difficulty found in attempts to inject them by piercing the capsule with the injection needle. The degenerations which are common in the tumors depend upon compression of their vessels and are caseous or fatty most commonly.

Myxoma and *enchondroma* have been found in the lymph nodes but malignant tumors are more common. *Primary sarcoma* is sometimes seen and differs from the "lymphosarcoma" mentioned above in its tendency to form metastases elsewhere than in lymph structures, while the other is confined to them at first. The sarcoma may be of any form but most often it is of the small round-celled variety; it usually starts in the fibrous structures of the ganglion, if fibro- or spindle-celled sarcoma. Melano- and angiosarcoma also occur, the former usually secondary. Of the secondary malignant tumors *carcinoma* is most frequent. Thus in carcinoma of the mammary gland the secondary manifestations in the ganglia of the axilla and the neck may appear very early and reach a large size even before the primary tumor reaches the skin and ulcerates. Examined early, the lymph nodes exhibit metastatic carcinoma cells among the normal elements of the part, though there is absolutely no gross evidence of participation; hence the importance of removing all adjacent lymph ganglia in operative procedures. In this case also the protective office of these nodes may be observed in the period of delay in metastasis in the early stages as neighboring groups become affected. (See Pt. I., pp. 170–1.)[1]

Infectious Granulomata. *Tuberculosis.*—In the local tuberculosis of the lymph nodes which is peculiarly marked in children before puberty, the infection most commonly occurs along the alimentary canal; perhaps from the use of infected milk, tubercular abscess in the udder of the cow being rather frequent and all the more dangerous because the animal's general condition does not suffer for a long time. Bacilli may pass into the milk even without disease of the udder.

The pathogenic action of bovine tubercle bacilli upon man has lately been denied. It must, however, be remembered that where it seems to appear as a practical observation the subject is usually an infant, whose lax tissues are less resistant than those of the adult. The infection, furthermore, is not simple. Colon bacilli and others probably combine in the process, after the normal epithelial defence has been weakened by fermentation in the intestine.

The cervical nodes are often infected, in many cases through the tonsil, bacilli lodging in the crypts of this lymphoid tissue and being carried to cervical ganglia by phagocytes; the mesenteric nodes are infected in a similar manner and less frequently the bronchial. The node involved swells to a moderate degree, becomes hyperemic, paler after a time when hyperplasia follows, and then the central portions become caseous. As in the lung and other parts of the body, mixed infection by the pyogenic organisms frequently follows and the cheesy gland becomes an abscess, adhesions to adjacent tissues with perforation in the line of least resistance may follow. As in the neck, this is usually by external evacuation and after a time the necrotic tissue is all extruded and stellate and irregular scars remain. If the pus is retained by a thick capsule it may dry and be infiltrated with lime salts. Secondary general infection is always possible from a lymph node where bacilli linger, especially when free entrance to the circulation is gained by adhesion with a vein and later perforation. Microscopically the lymph nodes present all stages of typical tubercular tissue and its degenerations, with bacilli in the giant cells or between the cells; after a time it is difficult to find bacilli in the ganglia, but either they or their spores must be present for injection of caseous or softened material from them into animals is usually successful.

Syphilis.—One of the first symptoms of constitutional infection is the indolent bubo; this may appear in either groin when the chancre is situated on the genitals, or in any corresponding node when extra-genital. The bubo is made up of nodes which are not very much swollen, but little painful or tender, and not given to suppuration; in these respects it differs from bubo following chancroid. In the secondary stage many or most of the superficial lymph ganglia are enlarged. After reaching a moderate development these nodal swellings remain unchanged for long periods in the absence of antisyphilitic treatment. Microscopically they present thickening of the stroma and of the vessel walls, increase in the epitheloid cells of the sinus and the spindle cells of the stroma. In tertiary stages gumma formation may occasionally occur in the lymph structures, but like all the severe forms of tertiary syphilis they are less and less frequent with the general tendency of the disease to run a milder course.

Leprosy causes fibrous hyperplasia, with fatty degeneration of the cellular elements, in the nodes corresponding to the skin lesion; later the internal nodes are also involved. *Glanders* produces swelling with small-celled infiltration.

Degeneration.—Simple atrophy is a constant accompaniment of senility, with cessation of the function. Fatty and amyloid degeneration occur at times, caseous changes with tuberculosis and syphilis, calcification as the final stage of these or after bone disease, as caries, with absorption.

Status Lymphaticus.—An unexplained relation exists between general lymphatic hyperplasia and certain sudden deaths, sometimes of adults but especially of children, and of great practical importance in connection with surgical anesthesia. Cases have been reported which had nothing to do with administration of an anesthetic, the child suddenly becoming cyanotic, with convulsions of the facial or trunk muscles, involuntary urination and defecation, and death in a few minutes. Perhaps the greater number, however, have occurred with the use of chloroform, often to but slight degrees of narcosis or sometimes afterward. On post-mortem examination the thymus is usually large, so that *thymic asthma* and *laryngospasm* were names formerly applied, but other lymphatic structures present similar gross appearances. The pharyngeal and faucial tonsils, the follicles at the base of the tongue, the lymphoid elements in spleen and intestine, and the systemic lymph nodes may all or most of them be enlarged. Associated aplasia of the aorta and entire arterial system, dilatation of the heart and general delicacy and imperfect development have been described. At times more than one member of a family has died in this condition in anesthesia or apart from it. But little can be said of its causation or its significance. Altered products of the hyperplastic tissues have been assumed, which induce convulsion and syncope on slight stimulation. The tendency appears to be to outgrow it, but during the first years of life clinical experience has proved the wisdom of examining all accessible lymphoid tissues before administering chloroform, and withholding it in the presence of general hyperplasia.

Other lymphatic structures, as the tonsil and bone marrow, remain to be treated. The tonsil will be considered in connection with the alimentary canal. So far as the marrow requires special mention here, apart from diseases of bones, it is chiefly as alterations in the blood produce lesions in the marrow or the converse of this. Finely divided irritating and infectious material tends to collect in the marrow as in the spleen and lymph ganglia, and hence osteomyelitis is occasionally a complication of scarlet and typhoid fevers, variola and measles, malarial and pyemic infections. In pernicious anemia red marrow forms where usually only fatty marrow is found, perhaps in consequence of vicarious function when other blood-making organs are diseased. Normally red marrow occurs in the bones of the skull and the pelvis, the thorax and the proximal ends of the arm and thigh bones; in the latter situation fat takes the place of nearly all of this with the advance of age and in late years it becomes mucoid. When pathological influences reproduce the fetal red marrow it occurs centrifugally in the long bones, contrary to the physiological replacement of red by fatty marrow. In course of this change the fat cells disappear with the increase of lymphoid cells with large nuclei, red cells often nucleated, microcytes and poikilocytes, eosinophiles, myelocytes and giant cells which may contain red cells. The gross appearance of the marrow may be red and translucent (so-called "raspberry jelly") as in pernicious anemia, or variously mottled from pink to gray or yellow and puriform or even greenish, as in leukemia. Beside the cells mentioned the microscope discovers Charcot-Neumann crystals and pigment. At times the other lymph structures are relatively but little involved, or late in the disease, and hence a myelogenous leukemia is spoken of, in contrast to the lymphatic and the lienal or splenic (see pp. 54–7).

The Adrenal Body.

Malformations are not common; they include absence with marked general defect, fusion by a bridge of similar tissue, supplementary adrenals connected with the solar and renal plexuses, and dispersion of fragments in the liver, kidney and genital organs. From these hypernephroma may develop.

Hemorrhage in constitutional conditions may involve one or both organs and occurs at times in the new-born.

Inflammation is unusual; it may be purulent and either diffuse or localized.

Tuberculous disease is one of the commonest lesions of the part and is often the chief pathological find in Addison's disease; but it occurs also without any trace of pigmentation of the surface. The tubercles may be miliary or in caseous foci, cause increase in the size of the organ, rounding of its sharp margins, and usually are secondary.

Syphilitic induration may occur in a congenital form and with gumma caseous degeneration may occur.

Addison's disease, supra-renal melasma, is a clinical complex of emaciation, anemia, muscular weakness and pigmentation of the skin and mucous membranes (mouth) of a peculiar metallic color, which has given the disease the name cutis aenea. The only pathological lesion in many cases is an adrenal lesion, which in some cases is absent, and, further, may occur independently of the other and general disease. Other pathological conditions which at times are associated are sclerosis of the ganglia of the abdominal sympathetic, dilatation of its vessels and necrosis of the ganglion cells; in other cases pigmentation and fatty changes in these and other parts of the sympathetic. Experimental removal produces hemorrhage in the spinal cord and degeneration of nerves in animals; pigmentation seldom occurs and then is not surely the effect of the operation. The function of the gland appears to be the regulation of vaso-motor conditions rather than metabolic processes and whether its lesions have a causal or necessary relation to pigmentation of the surface and cachexia is not wholly determined.

CHAPTER IV.

ALIMENTARY CANAL.

The Mouth.

Malformations.—The commonest form of congenital imperfection in the development of parts about the mouth is fissure in the upper jaw, which may involve only the lip (hare-lip) or the bones, including the roof of the mouth; nasal, buccal and pharyngeal cavities then form one chamber. Such a fissure usually follows the suture between the premaxillary bone and superior maxilla; it may occur on both sides. The tongue may be too large or too small, or, as a great rarity, double; the lower jaw may be lacking, the lips may be fused or absent.

Circulatory Disorders.—*Anemia* of the lips and mucous membrane is a well-known symptom of general anemia. *Congestion* occurs in inflammation and early in certain infectious diseases, often with an eruption of the characteristic superficial lesion, even before it appears on the skin. *Passive congestion* is part of general cyanosis in lung and heart disease. *Bleeding* is common in purpura, scurvy and similar diseases.

Inflammation.—The developmental identity between the skin and the lining of the mouth is seen in the great similarity between inflammation in the one and the other; often the lesion passes from skin to mouth or *vice versa.* The process may be associated with free desquamation of the epithelium and sympathetic excess of saliva; when the desquamation is severe shallow ulcers form. As in other superficial parts the inflammation may be simple and soon disappear, it may be more severe and accompanied with suppuration, or it may be chronic and result in fibrous hyperplasia and thickening of the part; a form of the latter is seen in ill-nourished children with nasal discharge, eczema of the upper lip and permanent increase in the thickness of the latter.

Acute inflammation of the entire mucous surface of the mouth is called *stomatitis* and may be of several kinds.

Acute catarrhal stomatitis follows irritation and injury from chemicals, hot fluids, with the eruption of the teeth and from excoriation by partly decayed teeth; in the latter form the trouble is more apt to be chronic, as is seen also in alcoholic dyspepsia. The desquamation may be slight or profuse: in later stages the epithelium proliferates without desquamating and forms areas of thick and white appearance, and at times the mucous glands enlarge and give a granular effect to the parts. The increased and altered saliva may deposit mineral salts on the teeth and in the edges of ulcers in severe attacks.

Aphthous stomatitis, thrush, is due to the growth of a fungus, oïdium albicans, through and between the superficial epithelia; the first stage is redness, then patches of white false membrane appear, attached firmly to the tissue beneath but later becoming loose. Beside the fungus and epithelial cells found in the membrane, many other forms of bacilli and cocci are present. From the anterior portion of the mouth cavity the fungus may spread to the pharynx and along the esophagus, invading parts covered with flat epithelium; less often where the cells are cylindrical. At times the parasite passes into the bronchi, and when there are superficial ulcerations on the skin these may also be invaded. The fungus may be cultivated on acid media and forms mycelial filaments when there is but little sugar present, with sugar the cells are chiefly round or oval and budding, like yeast (saccharomyces). It is improbable that the fungus can invade healthy epithelia; clinically it occurs only in the very debilitated, with neglected oral hygiene, and most commonly in young children. (See Pt. I., p. 202.)

Vesicular and *pustular inflammations* may occur in the mouth as on the skin, a good example of the latter being the pustules of variola and secondary syphilis, which at times occur in numbers on the soft palate and neighboring parts.

Scorbutus affects the mouth as one of the characteristic points of attack. It may be the first part invaded, beginning with swelling and dusky hue about the teeth, bleeding on slight cause or spontaneously; the teeth become loose, the gums recede and ulcers appear upon them; gangrene and entire loss of the teeth with necrosis of the bones may result.

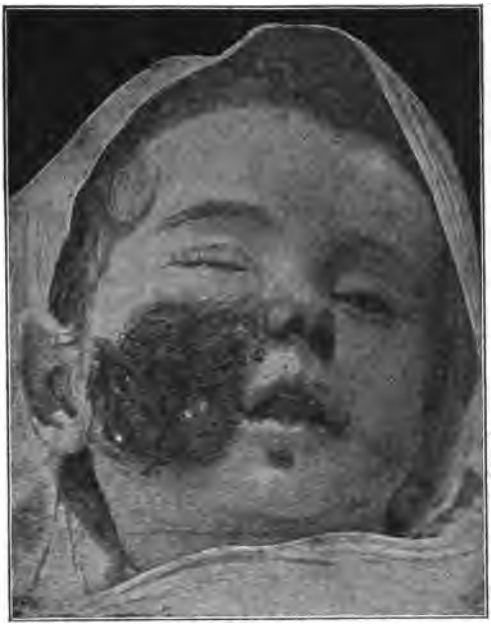
Mercurial stomatitis in persons with an idiosyncrasy may result from but trifling use of the drug; in others it comes on after long treatment with it. The teeth give pain when shut tight together, the saliva increases to many times the daily normal and has a foul odor, there is swelling in the cheeks and the gums, and here and along the tongue shallow ulcers form.

Tubercular invasion of the lips and tongue may occur as a primary lesion, as a papule with eroded surface and early formation of an ulcer with thickened edges; the color is at first yellowish, later the base of the ulcer is made of sluggish granulation tissue. The resemblance to epithelioma and softening gumma is so close that the tubercle bacilli should be found (most often in giant cells) before making a positive diagnosis.

Syphilis affects the mouth either as a primary lesion (chancre) on some part, as lip, tongue or tonsil; or as mucous patches and other secondary forms; or as gumma, most often in the corners of the mouth or on the palate; the gumma is apt to make severe ulcers as it softens and often perforates the velum or destroys the uvula.

Actinomyces is liable to invade parts about the mouth, but probably there must be some abrasion of the surface before it can cause important lesions; frequently through carious teeth the fungus reaches the alveolar border of the under jaw. Its course, wherever it settles, is either a rapid suppuration, which is not common, or a steady destruction of surrounding tissue with but little pus formation.

FIG. 13.



CASE OF NOMA. (*Stengel.*)

Gangrene of the Cheek, Noma.—Children debilitated from severe and chronic disease, in surroundings of dirt and imperfect supply of air and food, occasionally present a rapid form of buccal gangrene. It begins on the inside of either cheek as a small blister with dark and foul contents; the skin reddens and then turns darker, vesicles form on the outside, the mucous membrane is bluish and livid, and the entire area becomes necrotic. The process invades the tissues in all directions, no line of demarcation forms, the cheek is perforated, and as far as the eyelid and the edge of the lower jaw all the tissue is dead; usually but one side is involved and the tongue is apt to escape. The usual result is death; in rare cases the edges of the part granulate and heal with a large scar. Although most common in female children under seven, noma has been observed at all ages and in either sex.

Inflammation of the Tongue. *Glossitis.*—Beside attacks of this disorder when other parts of the mouth are involved, it occurs by itself either as a superficial or a deep parenchymatous inflammation. The first may be the sign of widespread digestive disorders, associated with functional and organic disease of the liver or any part of the intestinal canal, or it may follow local irritation as from sharp edges of carious teeth, tobacco, etc. A chronic form of epithelial hyperplasia, often very circumscribed, is known as psoriasis of the tongue; the affected areas are thick and white, lie along the edges or on the upper surface of the tongue, may spread and join, sometimes the abnormal epithelium is cast off and a small ulcer is left. The disease is important clinically because of secondary development of epithelioma on the site of the ulcer. Another form of superficial inflammation of the tongue is melano-glossia or black tongue, in which the filiform papillæ reach a remarkable size and give the upper surface a hairy appearance and a black color. The condition is explained as a hyperkeratosis with local increase of pigment, or as due to a fungus resembling oïdium albicans, which is almost always found mingled with the thickened epithelia and various bacterial forms. Phlegmonous inflammation of the deeper tissues of the tongue differs from other suppurations but little and is due to similar causes; the end of the process may be a permanent increase in size. Tubercular and syphilitic ulceration may occur alone on the tongue or when other parts of the mouth are also involved.

Tumors.—Almost any of the benign tumors of the connective tissue group may occur in the mouth and seldom reach a large size; the mucous glands about the lips may hypertrophy and their lining cells undergo either hyaline or colloid changes; nevi and cavernous angioma are both observed, most often on the upper lip. Among the malignant tumors epithelioma of lip and tongue is the most common. On the lip it usually comes on the under one and is laterally placed, affecting men almost exclusively. If superficial it ulcerates rapidly and soon invades the sublingual lymph nodes. On the tongue one or the other edge is marked by a grayish and dense area, slightly prominent, and soon afterward a rapidly spreading ulcer; while but one half of the tongue is usually affected the tumor spreads quickly to the gum, cheek or larynx, and because of lymphatic transportation its course is quick and return after operation frequent. Epulis is the name given to various tumors which start in the alveolar borders of the jaws; these may be benign fibromata, but most often are some form of sarcoma; giant cells are common in them. Osteosarcoma from the outer aspect of the teeth. Lower jaw may develop to enormous size.

The Teeth.

In number and size, variations of the teeth are common; supernumerary teeth and extra digits on hand or foot sometimes occur together in more than one of a family; when teeth of the first set remain too long the corresponding ones of the permanent series may be small or altogether absent. With congenital syphilis the incisors, especially the middle upper of the second set, are excavated along the cutting edge in crescent form and the others may be peg-shaped or irregular, but this and the yellow hue of teeth have been found where syphilis could not be proved.

Inflammation may affect the periosteum of the tooth's insertion or gain entrance to the pulp cavity; in either case suppuration may occur with death of the tooth. Chronic inflammation and caries of the teeth usually is due to destruction of the enamel by acid fermentation of particles of food left between the teeth; about sixteen kinds of bacteria living in the mouth may produce such fermentation. Through the destroyed enamel the tooth loses its mineral salts, bacteria invade the dentine tubules and the tooth becomes carious; this is the result of more than one kind of organism, a mixed infection, with cocci as the most numerous variety.

Palate and Tonsil.

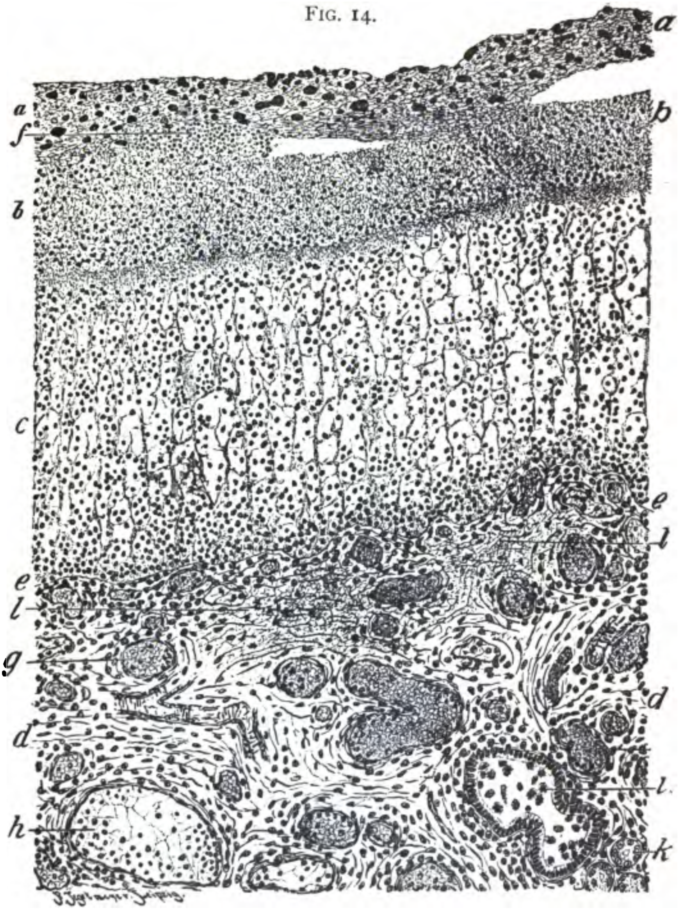
As in other situations, anemia and hyperemia follow such conditions in the body at large, acute congestion is seen in the first stage of inflammation, passive congestion with any condition of obstruction to the return circulation; in the latter case the veins of the part may be varicose. Hemorrhage may be caused by wounds, foreign bodies, and cachectic conditions; when the blood collects between the layers of the soft palate a large hematoma may form (staphylhematoma). Edema occurs with inflammation and ulceration and, because of the looseness of the tissue, may be very marked.

Inflammation.—The localization of the inflammation is diffuse, over the whole inner surface of the mouth and pharynx, or confined to the velum and uvula; it may be of any kind and is divided into symptomatic and idiopathic. The name given to these inflammations is *angina*, catarrhal, croupous, diphtheritic or phlegmonous according to its nature.

Catarrhal angina may be acute or chronic; in the former case it follows reflex or direct irritation and may be on the posterior surface of the velum and include the tonsil, or be variously circumscribed. The part is edematous and swollen, redder than normal, at times dotted over with swollen glands and these may be prominent from a drop of pus in each; such an appearance is common with the exanthemata. Vesicles may form, at times herpetic, and shallow ulcers from desquamation of edematous epithelia; when these latter cells are much swollen but remain attached they resemble the false membrane of diphtheria; under the microscope pus-cells are found inside the epithelia. In a chronic form such a simple angina may follow frequent attacks of the acute and a granular and varicose appearance is then characteristic.

Diphtheritic Angina.—Microscopically the croupous inflammations of the various mucous membranes are more superficial

FIG. 14.



DIPHTHERITIC INFLAMMATION OF THE UVULA. (*Ziegler.*)

***a, b, c,* Three layers of fibrin, the upper containing groups of cocci, the others holding leucocytes in their meshes; *d,* connective tissue infiltrated with cells; *e,* boundary zone of submucous tissue, infiltrated with round cells; *f,* red cells in groups; *g,* dilated blood-vessels; *h,* dilated lymphatic, holding serum, fibrin and pus-cells; *k,* transverse section of a gland tubule; *l,* fibrin in the connective tissue.**

than diphtheritic, the pseudo-membrane, made up of desquamated epithelia, pus-cells and fibrin, lying upon the membrane while in the diphtheritic inflammation the layers under the false membrane are necrotic, infiltrated with fibrin, and the resulting ulcer much deeper. Such inflammations in the palate occur after various injuries, chemical and other, and in the course of several acute diseases, scarlet fever, measles, variola and others. In all of these the exudative inflammatory products are mingled with pus and necrotic cells from the mucous surface. The characteristic lesion of diphtheria as a specific disease is a false membrane made up of two elements: fibrinous exudation, the fibrin lying between the cells of the part, dying or dead, and coagulation necrosis even into the depth of the mucous membrane, with well-marked emigration of white cells and to a less extent of red. Such a false membrane is most often observed on the tonsils and the pillars of the fauces, but it may invade the nasal cavity (through the pharynx), the larynx and bronchi, and even the esophagus and stomach. At first its color is white or gray, at times with a yellow tinge, but if red cells are present in numbers it may be dark; later it turns yellow or grayish-green. It may be expelled in bits by efforts of coughing and renewed many times in the course of the disease, and there may be little foramina in it corresponding to gland openings or projections which fitted into the lacunæ of the tonsil.

The amount and distribution of the membrane gives a suggestion of the gravity of the disease; in severe cases it forms rapidly and spreads into the pharynx and nose or downward. It is very often the fact in these severe cases that there is a mixed infection especially with streptococci, and hemorrhage and gangrene are most frequent when this is marked. On the edge of the affected spot the epithelia may persist although covered by the growing membrane, but directly under it they are lost, for the fibrin lies between them in the place of the cement substance and thus their attachment is soon loosened. Whether the coagulation of the fibrin here precedes or follows the necrosis has been disputed, but the probability is that necrosis follows coagulation. The attachment of the pseudomembrane is closer over flat epithelia than over cylindrical but in either case is apt to be cast off in fairly large pieces and the tissue under it ulcerates and heals with but little scar formation. The local swelling may be so great as to cause a stenosis.

When the lungs are also involved it is usually a lobular pneumonia with a descending croupous bronchitis or without participation of the bronchi. The lymph nodes of the region about the angle of the jaw are usually swollen in proportion to the severity of the attack. Ascending infection may pass through the Eustachian tube and set up otitis media.

Perhaps as important as the local infection there is constitutional disturbance of all gradations; limited to a slight febrile reaction, or with symptoms of profound septicemia. The postmortem examination in severe cases reveals marked hyperemia of the kidney, interstitial edema and bleeding about the glomeruli, and cloudy degeneration of the epithelia. Similar degeneration of the heart muscle occurs and may be the immediate cause of death, after the disease has apparently run its course. In the spleen hyperemia with swollen lymphoid elements may occur. Paralysis of the velum is common after diphtheria but the paralysis may affect distant muscles also, as those of the eyes, larynx and diaphragm; the lesion in the muscles is usually an inflammatory small-celled infiltration between the fibers with granular degeneration of the muscle cells; fatty changes follow rapidly. In the spinal cord hemorrhages and degeneration of the ganglion cells correspond and in some very severe cases there are multiple punctate hemorrhages in the membranes of the brain and in the cortex, as also in liver, kidney and alimentary mucous membrane.

The exciting cause of diphtheria is the bacillus diphtheriæ of Löffler, which is constant in the false membranes of palate, nose, pharynx and bronchi, and may be recovered from affected tissues even before the pseudo-membrane is well formed; and similarly may linger for weeks in the apparently cured or occur in healthy members of a family when attending a case. It is probable that the bacillus does not attack a healthy mucous surface, and its most damaging results are effected by association with streptococci. The bacillus is about 3–4 μ long and slightly thicker than a tubercle bacillus; the end may be apparently swollen and more deeply stained than the middle part, slight bending and variations in length are common. They stain readily with methylene-blue. At first the specimen obtained by swabbing the throat with sterile absorbent cotton may be practically pure culture; later the streptococci may outnumber the bacilli. While often confined to the mucous surfaces the bacilli may be recovered from the lymph ganglia of the part, the lung, kidney, heart, blood, spleen and brain. Probably the general symptoms are due not so much to transported bacilli as to toxin absorbed from the primary affection, for filtered cultures from broth reproduce them when injected into susceptible animals (dove, goat, guinea-pig).

When an animal has been dosed with increasing strengths of filtered culture, or with bacilli which at first are reduced in virulence, its serum becomes antitoxic, and when injected into a susceptible animal protects it against infection. More important for therapy, it is found that injection of such serum has a curative influence when the disease is beginning, most marked when given very early after infection. (Pt. I., p. 227.)

Diphtheria antitoxin is prepared in flat-bottomed sterile glass flasks; at first it was thought necessary to keep a current of moist air running through the flask but this is proven to be superfluous. Beef broth with 2 per cent. peptone is put in the flask and sterilized, inoculated with vigorous bacilli and put in the incubator at 37° C.; in three to four weeks enough toxin has formed. The broth is then filtered under strict precautions through a porcelain filter (Pasteur-Chamberlain), and kept in the dark in sterile, well-stoppered bottles. Guinea-pigs (average 300 gm.) die in 48–60 hours from the injection of 0.1 cc. of the filtrate.

To immunize, dilute the filtrate with 25 per cent. Gram's fluid, inject a rabbit with 0.5 cc. of the mixture; after the reaction has quite disappeared (slight fever, increased pulse), inject a similar amount with less iodine solution; a point is reached where the iodine is left out and the animal can take as much as 100 times the amount fatal to a non-immunized rabbit. Of all animals the horse has been found most satisfactory for practical use as it bears large doses, thus shortening the time, and furnishes large amounts of serum. The antitoxic serum is obtained by drawing blood from a vein by a cannula into sterile vessels under strict antisepsis; to preserve the serum camphor, tricresol, carbolic acid and other chemical antiseptics may be added, but with careful methods the serum is sterile and does not require such additions, which are undesirable.

The value of the antitoxic power of serum thus obtained is based upon the least dose of toxin which is fatal to a given weight of animal. Several guinea-pigs may be selected, as nearly of a size as convenient (about 250 gm.) and each receives an injection of toxin, the dose varying for each; by the fourth or fifth day some will have died, and the minimum dose of toxin is taken as that which will kill 250 gm. of guinea-pig in 4–5 days. This should be subjected to further trial on other animals till determined beyond doubt. The serum remains to be tested, and a unit of measurement established; this is done when it is learned how much serum will protect against the minimum fatal dose of toxin, *only it is stated as ten times the amount of antitoxin* which is effective *against ten times the minimum fatal dose of toxin.* Thus several doses of toxin are known to contain ten times the minimum fatal quantity, and to these varying amounts of antitoxic serum are added; to the first 0.01 cc., to the second 0.05 cc., to the third 0.1, to the fourth 1.0, and to another a higher amount. These mixtures of toxin and antitoxin, in which the latter element alone varies, are injected into guinea-pigs. That mixture is held to be effective which, with the lowest proportion of antitoxic serum, has prevented fever and local swelling; it is considered as one tenth of a unit. If this may have been the second of the preparations ten times the proportion of serum gives the antitoxin unit, in this case 0.5; in other words, that amount of serum contains one immunity unit of antitoxin serum. Three strengths usually occur in the shops, a weaker for preventive inoculation, a stronger for treatment (by hypodermic injection) when the case is seen early in the disease, and a stronger yet for cases seen after the first 24 hrs.; more than one injection may be required. When the serum acts favorably the local and the general symptoms soon begin to improve. It is but fair to add that the value of serum therapy is still contested by many careful writers, who contend that deaths from lung and kidney complications are more common under it and practically keep the mortality where it was before antitoxin treatment; and also that a large part of the apparent improvement since the introduction of antitoxin is to be explained by improved general hygiene and affects other diseases, not treated in this manner, to even a more marked degree than diphtheria.

The rate of diphtheria mortality before and after the use of antitoxin in several cities may be seen from the following table:

Locality.	Before Antitoxin. 1889–1894.		After Antitoxin. 1895–1900.	
	Cases.	Mortality %.	Cases.	Mortality %.
New York City.	35,944	28.03	62,002	14.32
Massachusetts.	13,332	28.30	28,470	15.60
Philadelphia.	19,828	31.07	21,429	27.02
London.	11,598	30.30	29,055	17.00
England.	64,189	27.50	68,488	19.80

Phlegmonous Angina.—The severity of the inflammation is marked by the great hyperemia and edema of the tissues and the infiltration by pus-cells. The tonsils may so increase in size as to block the entrance of the pharynx, and become so painful that the jaws are held rigidly shut. Spread of the inflammation may cause fatal edema of the glottis. Various bacteria may produce such conditions, among them streptococcus and staphylococcus, pneumococcus, bac. mallei, and anthrax. Abscesses may form and discharge outwardly or into larynx or esophagus, and the affected parts may become gangrenous. Septicemia is to be expected and may be the cause of death.

Tonsillitis.—In any of the forms of inflammation about the palate the tonsils may be implicated and bear a large share of the damage, but frequently they are the only site of the lesion and several forms of *tonsillitis* are therefore described, which differ pathologically but little from those already mentioned. *Catarrhal, lacunar* or *follicular,* and *phlegmonous* tonsillitis are the usual forms. Follicular inflammation may invade but a few of the crypts and remain at a low development for long periods, and as the bacterial elements present may be virulent, this bears a clinically important relation to diphtheria and tuberculosis. When the bottom of the crypt is weakened and perforated by the inflammatory process the severer forms of phlegmonous tonsillitis occur, one or both of these organs will be involved, considerable constitutional disturbance is usual, abscesses may form and break in varying directions and are most dangerous when involving the vessels of the neck or opening into the larynx. A *chronic productive form* of tonsillitis results in great increase in the size of the part, and as the condition is often found in children, it is of great importance for their nutrition, for they are deprived of their normal supply of oxygen at every intake through the narrowed air passages, particularly so because they are apt to form the habit of mouth breathing, and their digestion suffers also. In these cases there may be simple lymphoid hyperplasia, as also in the vault of the pharynx, differing from the fibrosis which follows repeated acute tonsillitis.

Tuberculosis may affect the tonsil primarily and become generalized from here by way of the lymph channels to the lungs. *Syphilis* is found as primary lesion, secondary syphilide and gumma. *Lupus* occasionally is transmitted from the outer surface. *Glanders* nodules and leprosy tubercles are also found in palate and tonsil.

Tumors.—Lymphatic hyperplasia is common in the tonsil, either as primary or secondary lymphoma. Chronic inflammation of the mucous membrane sometimes results in polypoid formations. Papilloma, myoma and fibroma are sometimes found, and, among the malignant tumors, sarcoma and extensions of epithelioma.

Pharynx and Esophagus.

Malformations are not common, and perhaps fistulæ from imperfect closure of a branchial cleft are the only ones of practical interest; it may be the second, third or fourth cleft, and the outer opening varies correspondingly. The fistula is complete between pharynx and external world or either end is closed, and such diverticula from the pharynx may be large and cystic. *Disorders in blood supply* follow those of neighboring parts and do not require special mention.

Inflammation follows injuries and extension from other parts, and may be of any variety, perhaps most common is the *chronic pharyngitis* of smokers and those who use the voice much, the mucous membrane being dry and granular, secretion scanty and viscid, vessels often varicose. Of the deeper inflammations *retropharyngeal abscess* is important, from caries of the cervical vertebræ most commonly, due to infection with tubercle bacilli. The end of the process varies with the acute or chronic nature of the abscess formation. The more rapidly formed are apt to bulge forward into the pharynx and rupture while the slowly formed tend to follow fascial planes along the neck and perforate into the esophagus or bronchi or posterior mediastinum.

Inflammation of the esophagus follows swallowing of foreign bodies, hot fluids, acids and alkalies, and consists in some cases of hyperemia and desquamation, increased secretion being not demonstrable. In *chronic forms*, as in alcoholic patients, the mucous membrane is apt to be swollen and pigmented, of a brownish or grayish color, with small ulcers here and there; hypertrophy of the muscular layer, especially about the lower end, is often marked. *Suppurative inflammation* may come from the tearing and pressure of large foreign bodies or the ingestion of chemicals, as in attempted suicide, and the process may spread downward, dissecting between the coats of the tube.

As in the pharynx, *diphtheritic processes* occasionally spread from similar lesions in the mouth and nose. *Variola* produces its characteristic pustules and *thrush* spreads from the mouth.

Changes in the lumen of the esophagus are of great importance clinically from interference with the function of the part. There may be uniform dilatation when the cardiac orifice is stenosed, increased in many cases by diaphragmatic action about the passage of the tube through it, the section just above becoming first involved; the muscular layers usually show well-developed hypertrophy in this condition. *Sacculated diverticula* are more important. If they result from pressure upon the wall from within they are called *pulsion diverticula;* if from adhesions and consequent pulling from without they are known as *traction diverticula.* The first is more common in the upper part of the tube after loss of power in the muscular coat and hernial protrusion of the mucous. The usual seat is the posterior wall at the pharyngeal junction, owing to the thinness of the muscularis mucosæ in this region and the horizontal arrangement of the fibers. In form they resemble an oriole's nest, their lower portion being dilated and holding decomposed food, etc., communicating above by a narrow opening with the esophagus; the pressure of the dilated part may completely close the tube itself.

Traction diverticula are commonest about the bifurcation of the trachea from adhesions of inflamed bronchial lymph nodes and consequent contractions of the new-formed tissue. The shape of the dilatation is conical, the apex lateral from the esophagus, and by hard particles of food or ulceration in the apex perforation may occur and a cavity form in the tissue, containing decomposed food and pus; this may break into adjacent air passages and cause gangrene of the lung or into vessels with fatal hemorrhage.

Perforation and *rupture* of the esophagus may follow internal ulceration, and a peculiar form of this is found over the cricoid cartilage in states of extreme depression, or from external pressure by aneurysms and abscesses. Rupture has been described as the result of traumatism, or spontaneous. Regurgitation of gastric contents may partially digest the lower end of the tube and lead to perforation; as this may occur post mortem care is needed in its interpretation.

Tubercular, syphilitic and *typhoid* ulcers have been described in the esophagus, but are unusual; their healing may lead to local stenosis.

Tumors of several kinds are observed here, but the most important is carcinoma, which especially affects men of alcoholic habits. The new growth is found in the lower or middle third, most often at their junction with the left bronchus. The tumor is usually disposed about the whole inner side as a ring and may not attain a great perpendicular extent. First the mucous coat is destroyed and appears elevated in papillary projections, with stenosis of the lumen; then the process involves the outer layers of the part and adjacent tissues, partly directly and partly through the lymph nodes. To this are added dilatation about the stricture, collection of food, ulceration and at times perforation. Metastasis is common in pleura, lung, and liver.

Stomach.

Malformations.—The organ may be absent in non-viable infants, as small as the duodenum, contracted about the middle and hour-glass in form, or atresic at the pylorus. Various hernias may contain part of the stomach or all of it.

Circulatory Disorders.—The vessels of the stomach lie between the various coats of the organ, the arteries piercing the muscular layer at an acute angle, the veins being thin-walled and having no perivascular connective tissue, anastomoses being free and total supply of blood large to correspond with sudden functional demands; hence certain forms of variation in the blood contents of the organ are easily produced. *Anemia* with atrophy of the mucous surface occurs in long-standing anemic conditions and old age. *Hyperemia* is active and general (functional) during digestion, acute and distributed irregularly as the result of irritation ; especially marked on the summits of the rugæ in the pyloric region. *Passive hyperemia* occurs with liver and heart disease and submucous punctate bleeding is then not uncommon. Similar small *hemorrhages* occur in many cadavers as the result of vomiting and other convulsive movements during the last minutes of life. If such a point of hemorrhage is examined by a side light a marked loss of substance in the epithelia over it may be made out. Central nervous lesions sometimes cause such small hemorrhages in the glandular layer, and also certain cachexiæ, as hemophilia and scurvy. *Massive bleeding* from the mucous membrane of the stomach occurs with ulceration, especially the round or perforating ulcer (*ulcus rotundum*) and gastric carcinoma. Rupture of passively congested veins is observed in cirrhosis of the liver. *Melena neonatorum* follows imperfect respiration and consequent backing up of the blood through the right side of the heart, vena cava and portal territory.

Inflammation.—*Acute gastritis* occurs from irritation of various kinds and is commonly located in the pyloric end. If the irritant is a poison it may float over the food in the fundus and attack the pyloric region and duodenum without leaving any marked trace about the cardiac end, but taken on an empty stomach it may produce its effects in the fundus also. The mucous membrane is red and swollen, its secretion is increased and small bleedings under and in it are common. Convulsive contraction of the muscle layers may produce hour-glass forms. *Chronic inflammation* also is most evident in the pyloric end and here the mucous membrane may be prominently marked off into little polygonal areas, giving the surface a mammillated appearance ; the mucous surface is often covered by a layer of thick mucus more or less firmly adherent. As a result of chronic inflammatory hyperplasia the mucous glands may atrophy and the mucous layer be increased in thickness and pigmented. If the hyperplasia is localized one or several polypoid growths may reach a large size. All combinations of glandular, fibrous and polypoid hypertrophy occur, but in some cases the glands are chiefly atrophied and the new growth is fibrous, a condition known as *interstitial gastritis*. When the fibrous tissue reaches the state of contraction the mucous membrane may no longer be movable upon its supporting tissue and the lumen of the stomach is lessened, at the pylorus actually stenosed.

A *phlegmonous* form of *gastritis* occasionally results from injuries, chemical corrosion and progress of ulceration in the mucosa; then the submucous tissue and the intermuscular connective tissue are very edematous and later infiltrated with pus-cells. When this proceeds to abscess formation and involves the major part of the organ the mucous layer seems to float on a purulent collection under it, or here and there it is necrotic and undermined and the lesion is more restricted. Malignant endocarditis and pyemia cause multiple *embolic ulcers* in the stomach walls, anthrax produces regional necroses with hemorrhage, and occasionally small-pox pustules are found. In rare cases diphtheritic inflammation has occurred with the same lesion higher up or in the course of severe infectious disease, as puerperal fever and variola.

Round Ulcer of the Stomach.—This is sometimes called *perforating ulcer* and sometimes *peptic ulcer* of the stomach. It differs from ulceration of other mucous surfaces and many explanations have been offered to account for its formation. One idea is that thrombosis occurs in a vessel and as a consequence a small area of mucous membrane undergoes necrosis, this necrosed portion is no longer able to resist the action of the gastric juice and is digested off, leaving an oblique, cone-shaped loss of substance, most marked in the mucous layer and gradually decreasing to the serosa. At the same time, if there were necrosis of the mucous surface and the dead tissue were being digested, the process would undoubtedly lead to formation of a thrombus in the vein below. Embolism and spastic contraction have been supposed to occur in the arteries, but as to the former the stomach arteries are not end arteries but anastomose freely and such ulcers of the stomach do not occur specially often in embolic diseases, as endocarditis. Small hemorrhages in the mucous membrane from violent emesis may also cause the local death of the cells which permits the gastric juice to digest them, and possibly very hot drink may sometimes act in a similar way. The outline of the ulcer is usually round or pulled into an oval by the muscles, and when it perforates the serous coat the foramen looks cleanly punched out; the floor of it otherwise is pale and smooth, as are the edges also, but both may be swollen and firm. Such ulcers are usually small and single and situated on the posterior wall near the lesser curvature about the middle of the distance between cardiac and pyloric ends, but they may occur in any part and may be multiple and large. At times the ulceration opens a vessel of some size in its floor and fatal hemorrhage occurs. If the process heals the scar may contract and distort the size and shape of the organ; stenosis is most probable when this occurs at the pylorus. At times the serous coat is protected by adhesion to neighboring parts and then perforation occurs into the substance of adjacent organs or into hollow cavities, as the pleura, the transverse colon, or the peritoneal cavity, which latter may be fatal at once or after peritonitis. Gastric ulcer affects women more than men (about 75 per cent.) and before middle life. Cancer of the stomach sometimes develops from it.

Degeneration.—*Atrophy* results from chronic inflammation, affecting the glands principally, or occurs in pernicious anemia and old age. *Fatty degeneration* of the epithelial cells is one result of phosphorus-poisoning and is seen in some infectious diseases. *Amyloid* changes affect the stomach both in its mucous and its muscular layers, beginning, as so commonly, in the walls of the smaller arteries. *Pigment* is deposited in the mucous surface after hemorrhages and in the course of chronic malaria, and little areas of *calcareous* infiltration are sometimes seen with bone diseases.

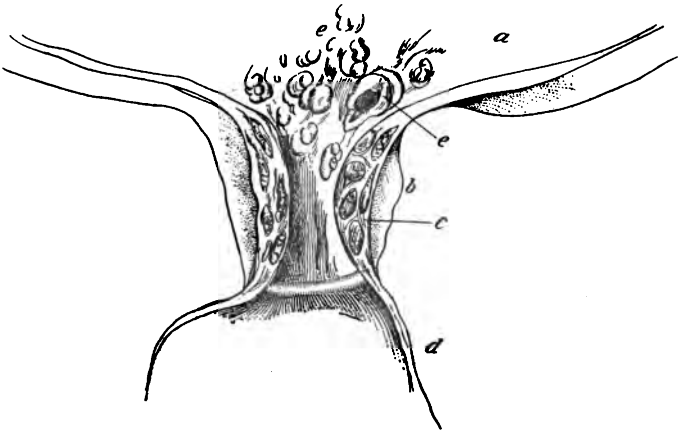
Gastromalacia is always a post-mortem change, though in the older writings it is supposed to occur during life when the digestive power of the gastric juice is specially high and vaso-motor action disturbs the blood supply. The softening of the digestive wall corresponds to the position of the body and is marked if the stomach is full of food and in warm weather: the appearance of the part varies with the amount of blood in the stomach wall from a pale gelatinous to a dark brown, the former especially in children and the latter in adults. There is often perforation and the wall of the foramen shows not the slightest trace of inflammatory reaction.

Infectious Granulomata.—*Tuberculosis* of the mucous surface of the stomach is almost unknown, though fatty and other degenerations of its glands with tubercular disease elsewhere is quite common; from the peritoneal surface it may, in rare cases, be affected. *Syphilis* occurs in a congenital form, at times associated with bleeding from the mucosa; gumma of the liver may produce stasis in the stomach veins or the latter may show gummatous infiltration. Occasionally in syphilitics a fibrous hyperplasia occurs all through the coats of the organ, and, by resulting contractions, the shape of it is much distorted. Rare cases of *actinomycosis* and *glanders* of the stomach have been reported.

Tumors.—None of the benign tumors are of much importance in the stomach, though fibroma and myoma may at times occur; sarcoma is a most unusual find; when present it may be either spindle-celled or fibro-sarcoma. Polypoid tumors of the mucous surface occasionally follow chronic inflammation and the larger ones are at times cystic. *Adenoma* occurs as a pedicled tumor, or diffusely spread through the wall; its mucous surface is apt to ulcerate. If typical adenoma, it is made up of glandular acini, with the membrana propria well preserved and usually but one layer of glandular epithelium covering it; transitional forms to carcinoma are apt to be found with adenoma, and then with the still typical glands are mingled others which show one or more layers of cylindrical epithelium on their walls, but the lumen is filled with a typical epithelia. Such a tumor forms metastases, sometimes rapidly, in the lymph nodes about the smaller curvature or the omentum or in more distant organs; it is usually called *adenoma destruens* or adeno-carcinoma, and very often undergoes colloid degeneration. (Vol. I., p. 166.)

Primary carcinoma of the gastric mucosa is commoner in men than in women, on the anterior rather than the posterior wall, and after middle life. In all these points it differs from gastric ulcer, though it may take its origin from a healed ulcer (3 per cent. of all cases). It appears in several forms and in the gross is either annular, often surrounding the pylorus, or parietal and localized, when a region of the wall is very thick and presents an ulcer

FIG. 15.



SCIRRHOUS CANCER AT THE PYLORUS. (After *Orth.*)

Thickening of the walls and stenosis, dilatation of the stomach, ulceration of the neoplasm. *a*, the dilated stomach; *b*, the thickened pylorus; *c*, its wall cut through; *d*, the duodenum; *e*, nodules of neoplasm.

with raised firm edges, or it may be diffuse and involve half or the greater part of the organ. It is usually medullary, or alveolar, colloid or scirrhous, the first of these growing very rapidly and with much ulceration, the latter more scar-like and slower in development. The ulcer is produced by necrosis of the epithelia as the result of locally altered nutrition, and while its thickened wall rapidly spreads in every direction its inner aspect continually breaks down; thus a large part of the mucous surface of the organ may be destroyed, large vessels opened and fatal hemorrhage produced. Less extensive ulceration may occur with the scirrhous form, and as the outline of this is usually round and its edges firm it may be easily mistaken for an ulcus rotundum; which fact together with the rather frequent development of carcinoma from a previous ulcer, makes the gross diagnosis a matter of difficulty in some cases. In the diffuse form, usually scirrhous but sometimes colloid, the whole stomach wall is thick, its rugæ are permanently infiltrated and stretched flat, its surface is consequently smooth, pale and cicatricial. Microscopically much of the muscle coat is replaced by fibrous tissue and here and there are small alveoli containing what is left of the atrophic epithelia.

Almost all varieties of cancer begin in the mucosa, but they soon attack the submucosa and spread in all directions under the apparently unchanged mucous layer. Soon the muscular layer is passed and the serosa involved, with nodular and projecting masses of neoplasm. From here the whole peritoneum may become invaded, as is common in colloid cancer. Other forms of the neoplasm tend to perforate adjacent viscera, the pancreas, liver and colon especially; the adhesions between the stomach and surrounding parts may prevent escape of stomach contents, or perforation may make fistulous tracts communicating with the pleura or the transverse colon, the esophagus when the tumor is higher up or the duodenum when at the pyloric end. The lymph nodes about the site of the lesion are early involved, but others at a distance, as in the supra-clavicular region, may also enlarge. A third manner of spreading is found in the veins. These may be opened on the surface of the ulcer and carcinomatous thrombi form in them; bits from these are carried to the liver capillaries and wherever they lodge a secondary tumor starts which reproduces the original form of the gastric tumor. Such metastases may be so rapid and reach so large a size that they entirely mask the still circumscribed gastric tumor.

Gastric Dilatation.—While stenosis of the pylorus may result in gastrectasia it sometimes occurs while the pyloric ring is well preserved. When the muscular elements in the pyloric wall are hypertrophied, with some degree of fibrosis also, a benign stenosis occurs; the orifice may be narrowed by scars of ulcers, or by cancerous new growths. The dilatation may carry the greater curvature downward, even as far as the pubic arch, while the smaller remains about normally placed, or the latter may also descend; in this case the change in the position of the stomach is most marked—it may lie almost perpendicularly, the pylorus in the right iliac fossa or even in the pelvis. When slowly developed a hypertrophy of the muscular coat usually co-exists; sooner or later, however, the distention mechanically thins the stomach wall and atrophy hastens the change. A form of dilatation from traction occurs, but seldom reaches so marked a degree, when the omentum is included in a hernia and constantly pulls on the greater curvature, or when the transverse colon is constantly loaded with feces in the habitually constipated, and also exerts a downward traction. When stenosis is the cause the dilatation may be called mechanical; when loss of tone and obscure nervous influences, together with atrophy of the muscular layers, cause the distention, it is termed dynamic. Stenosis occurs after cicatricial contraction of large sections of the stomach wall and from starvation. When the suspensory ligaments of the liver and other viscera become stretched and a general enteroptosis occurs, the stomach as a whole may be variously dislocated.

Foreign bodies of all kinds are swallowed by the hysterical and insane. Intestinal parasites occasionally make their way into the stomach, as round worms and tenia, and may be vomited. The contents of the stomach at autopsy may be food or medicine, or small amounts of mucus, or chemicals taken by accident or with suicidal intent; at times in intestinal obstruction, and in the insane from coprophagy, the stomach contains feces.

The microörganisms found in the stomach are yeast cells, sarcina, oïdium albicans in aphthæ, bacillus filiformis (especially with carcinoma), and others connected with fermentative processes.

The Intestines.

Malformations. — Congenital absence and reduplication of parts of the intestinal tract occasionally occur; stenosis is more common and may affect any section of the tract; the colon may end blindly at the sigmoid flexure or the rectum at the anus (*atresia ani*). The rectum also may be one common cavity with the urinary and genital organs (*cloaca* formation). The commonest diverticula arise from the omphalo-mesenteric duct and the flow of intestinal contents may be diverted out through the navel when this false communication is ample, the gut below being stenosed or atresic. Most commonly only a small diverticulum (Meckel's) like the thumb of a glove, is found above the ileo-cecal valve (in the new-born 0.5 meter, in the adult usually about 1.0 meter above), which projects from the convex side of the gut and is empty, its walls being formed as are the intestinal. If very long it may strangle part of the intestine or foreign bodies in it may lead to perforation.

Among variations in the location of the gut partial dislocations as herniæ may be mentioned. These are protrusions of the abdominal contents through some weakened or deficient portion of the enclosing walls, and they may be congenital or acquired. If the hernial sac lies without the abdomen it is called an external hernia, if the hernia passes through the diaphragm or into the retroperitoneal tissue it forms an internal hernia; the contents of the sac may include almost any one of the abdominal organs or be limited to a portion of the small intestine. The mechanism of all hernias may be referred to one of three causes, either increase of the intra-abdominal pressure in combination with a weak place in the wall, or abnormal length of the mesentery with the above, or masses of fatty tissue localized in the subperitoneal layer which mechanically or by atrophy increase the spaces between the fibrous elements, thus a pocket might possibly form into which abdominal contents might be pushed. Practically the only cause requiring consideration is the first, combined pressure and a weak wall, and certain parts of the wall are very liable to give way before the pressure of coughing, crying and muscular effort, especially lifting heavy weights. These are the inguinal and femoral rings, the neighborhood of the umbilicus, and parts of the diaphragm when imperfectly developed. Hernias through the sciatic foramen, the fibers of the levator ani, the obturator foramen and Douglas' pouch into the vagina have been described but are very uncommon.

The contents of the hernial sac, being exposed to circulatory disorders on account of the stretching and twisting of its vessels which may follow, are liable to various chronic inflammations which form adhesions between them; acute inflammation may occur after injury. Compression of the veins leads to stasis, thrombosis and gangrene; when the arteries also are compressed it leads to rapid edema, inflammation, hemorrhage and gangrene. Hernias which may be pushed back through the opening or ring by which they escape are called reducible; if adhesions or other cause prevent this they are called irreducible; when the neck of the sac is tight and stasis occurs the hernia is said to be strangulated. This strangulation may occur because of the small size of the ring, because intestinal onflow or external edema increases the size of the sac's contents, and hence the pressure on enclosing parts, or because other abdominal contents force their way in, as a piece of the omentum. If the strangulation is soon relieved and moist heat applied the gut may return to its normal condition. The part most often found in the sac is the small intestine, then the colon, and, with either, part of the omentum.

Stenosis and Dilatation.—The contraction of fibrous tissue after ulceration in the gut, or pressure from without, may narrow its caliber or occlude it; the part above then dilates while the distal portion shrinks and atrophies. Complete occlusion is rapidly fatal; degrees of stenosis not incompatible with life may cause gaseous distention and intestinal dyspepsia, with constipation. When the gut is twisted on its long axis, which may occur in any part not too firmly attached, and which is favored by a long mesentery, *volvulus* occurs. A second kind of volvulus occurs when a triangle of the mesentery, with a coil of the gut along its outer arc, twists about its apex, at the vertebral attachment. In either case, stagnation of the intestinal flow dilates the upper portion and the circulation being more or less completely stopped, thrombosis, with consequent edema and gangrene, is easily produced; above the constriction the gut may ulcerate and perforation occur. Adhesions between coils of the intestine in very favorable cases may allow perforation of adjacent walls. This reconstitutes the intestinal lumen and relieves the stasis in its contents; otherwise, fatal peritonitis is most probable.

When a portion of the intestine is received within the lumen of a portion below it, *intussusception* or *invagination* occurs, the receiving part being called the *intussuscipiens* and the swallowed portion the *intussusceptum*. The length of the latter may be a few cm., or the major portion of the gut above the site of the invagination; it grows by steady ascent of the receiving portion, so that what was the apex of the intussusceptum remains the apex however long it becomes. The inner portion may undergo a second intussusception, or several such places may be found along the course of the gut. Such a multiple invagination occurs in infants during the death agony at times and is without significance as a cause of death. When an invagination is formed it is almost always in the direction of the intestinal flow, the upper part being received into the lower, and its origin, while obscure, may be in convulsive peristalsis; in rare cases reverse peristalsis occasions an invagination in the other direction. Because of the tension and twisting of the mesentery of the intussusceptum circulatory disorders immediately follow, with edema and stasis inside and inflammation without; the result of the inflammation is to bind the peritoneal surfaces together and make the dislocation irreducible. The part most often invaginated is the lower end of the ileum with the head of the colon into the colon in adults, the ileum into the cecum or the descending colon into the sigmoid portion in children. The end of the process varies with the degree of disturbance in the blood supply and stenosis in the gut. It may become permanent, with the fluid contents of the gut passing fairly well; the intussuscepted portion may slough into the canal and be carried away, with nearly complete cure of the condition; or fecal obstruction may be complete and death rapidly follows; or perforation may set up a fatal peritonitis. Some cases appear to result from polypoid tumors within the intestine, which, by peristalsis, drag on a portion of the wall and invert it; others occur with large masses of imperfectly digested food in the intestine, and probably irregular peristalsis; many others can not be explained.

The rectum is liable to prolapse when its walls are dilated and loosely attached and when the sphincter is weakened; it usually occurs with violent efforts of defecation and when the prolapsed gut is replaced it is very apt to protrude again. If left unreduced it may become inflamed acutely, or even gangrenous, the sphincter strangulating it more or less, or a chronic thickening is observed and the mucous surface is apt to be ulcerated in places.

Perforation and Rupture.—The intestine may be opened by perforating wounds, stabs and bullets most commonly, or by ulcerative processes from within. If the perforation is small it may be closed by fibrin and healed, if larger the intestinal contents usually escape into the peritoneum and cause fatal inflammation. When the perforation has occurred slowly, as in tubercular ulcers of the gut, fibrinous adhesions may prevent the escape of feces for long periods, and in some cases the ulcerative process opens through the abdominal walls and a fecal fistula is the result. Rupture of the intestine may follow contusions of the abdominal wall or enormous accumulation of gas in the canal. The most frequent place for ulceration and rupture is the appendix vermiformis, that is, apart from typhoid and other ulcers.

Disordered Circulation.—*Anemia* of the intestine occurs in starvation and other cachexias and after severe hemorrhage. *Acute congestion* may be localized about the lymphoid follicles or in scattered areas or diffuse, and the serous surface may share in it. *Passive congestion* is a common result of obstacle to the portal flow, as in cirrhosis and tumors of the liver, and to a less marked extent follows lung and heart disease with impeded venous return. In this case the veins of the gut and the mesentery are swollen and prominent, and a special line of varicosities sometimes develops along the reflection of the peritoneum from viscera to wall in long-standing liver disease with portal stasis.

Submucous *hemorrhages*, as little points, occur in the upper part of the small intestine with severe congestion and in hemorrhagic constitutional conditions; larger bleeding into the canal may follow ulceration, chemical corrosion, and wounds, as the bites of parasites, but sometimes it occurs without apparent cause; perhaps the commonest ulcer to cause intestinal hemorrhage is the typhoid.

Edema of the intestinal wall may be most marked on the mucous surface or general throughout the wall; it occurs with venous stasis, acute inflammations, and especially in specific inflammations as from anthrax infection. Embolism may produce infarcts but usually the profuse anastomoses between the vessels relieve the part. Certain forms of ulcer may depend upon such embolism and consequent necrosis of the mucosa (as in the duodenum after superficial burns).

Inflammation.—Simple acute inflammation of catarrhal form is common after causes which produce it elsewhere ; it may be reflex from chilling of the body, especially of the abdomen and buttocks, and irritants swallowed with food, and parasites, are common causes. The inflammation may be limited to one portion of the small intestine or involve the whole gut. The follicles are swollen and prominent on a hyperemic mucous membrane and rapid transudation into the canal, with increased peristalsis, hastens the passage of feces ; hence diarrhea is common in even slight grades of this disease. When large masses of epithelium are loosened from the wall and passed in the feces, it is called *desquamative enteritis*, and when fibrin converts sections of this into tubes it is called *croupous enteritis*, a form common with young children. In these severe forms erosion of the mucosa produces a few or many superficial ulcers, usually small and of a yellowish color from purulent infiltration. Sometimes the inflammation specially affects the follicles and very small ulcers remain, which may be lightly pigmented, singly or over large areas.

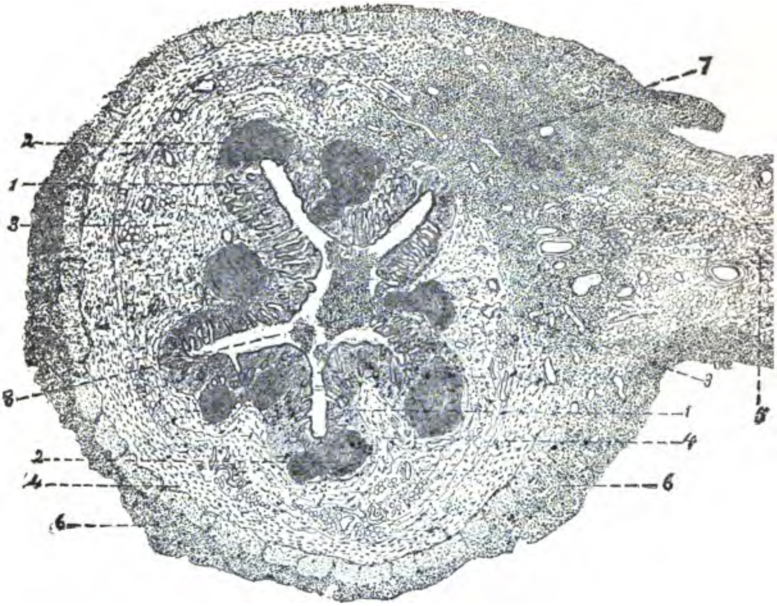
Chronic inflammation may proceed from an acute attack or repetitions of it, or may be caused by parasites and fermentation in the intestine. Certain slow poisonings, as lead and mercury and arsenic, regularly produce chronic catarrhal enteritis. The mucosa is thickened, covered with viscid mucus; passive hyperemia may give a cyanotic color to the whole thickness of the wall. Hyperplasia may affect the glands or the fibrous elements, or both, and the gross and microscopic appearances vary accordingly. Atrophy of the glands usually follows, and the site may be marked by pigment of a grayish or black color. The muscular coat may hypertrophy or atrophy. Functionally, constipation, or alternate constipation and diarrhea, with putrefaction of contents and symptoms of absorption of the products of decay—headache, dizziness, foul breath, coated tongue, etc., are the clinical manifestations of chronic enteritis, and the urine contains indican and ethereal sulphates.

Degeneration.—Atrophy and pigmentation have been mentioned. A marked form of the latter occurs in chronic alcoholism, in malaria and anthracosis ; the pigment may be found also in the liver and spleen. Amyloid degeneration of marked degree in other organs affects the intestine also ; independently it does not seem to occur. The amyloid material is found chiefly in the smaller arteries and capillaries of the villi and connective tissue.

While these characters of inflammation and degeneration are common to the whole tract of intestine, certain portions of it present peculiarities which may be briefly mentioned. Duodenitis usually follows gastritis and from the same causes. Because of the entrance of the common bile-duct in this portion, edema and ascending inflammation may close off the flow of bile and pancreatic juice, and hence icterus is a common symptom of duodenal catarrh. The cecum is specially liable to contain masses of feces, because of the mechanical disadvantage under which they pass to the transverse colon ; they may set up acute or chronic inflammation or lie passively and collect to enormous masses.

The appendix may contain hardened feces, calculi made up of stratified lime salts, foreign bodies as seeds ; and because of its narrow lumen and large proportion of lymphoid tissue and liability to hyperemia and edema, constriction of its vessels and acute inflammation readily follow. Slight wounds and necroses in its mucous membrane permit the colon bacillus or other pyogenic bacteria to attack its tissue and set up acute suppuration and as in other lymphoid areas (Peyer's patches) phagocytes may introduce the bacteria deeply enough to cause the inflammation. The bacilli and their products cause a round-celled infiltration with serous exudate ; in these inflammatory products as a culture medium they multiply rapidly, and they and their toxins are confined there ; the cells of the place break down, polynuclear leucocytes assemble in numbers, and a suppurative focus is established. When the appendix lies behind the colon the resulting abscess lifts this part upward; when it lies free in the peritoneum the adjacent coils of intestine adhere and shut off the inflammatory process from the general cavity. In these ways local collections of pus form, intestinal contents may be added, rupture may occur into the gut or the peritoneum or externally. Fatal peritonitis is the usual result in neglected cases. When the inflammation is less acute and more productive the lumen of the appendix may be obliterated.

FIG. 16.



ACUTE SUPPURATIVE APPENDICITIS. (*Delafield & Prudden.*)

Appendix removed by operation twelve hours after first symptoms. Streptococcus in the exudate. 1, mucosa of appendix; 2, lymphatic nodules in mucosa; 3, submucosa; 4, muscularis; 5, mesentery of appendix; 6, pus and fibrin covering the appendix; 7, dense infiltration of wall with pus.

Colitis may be limited to the cecum, the sigmoid flexure or any other part of the large intestine or involve the whole of it. Acute forms occur as dysentery and may be caused by poisons from without the body (mercury especially) or from within (as in chronic nephritis). Chronic forms are common with habitual constipation and the constitutional results of absorption vary from malaise and chronic headache to severe copremia and death in coma. When the rectum is the seat of the inflammation it is called proctitis, acute or chronic, and the former is very apt to pass over into the latter; the pathological appearances do not require special description. One form of chronic colitis is peculiarly resistent and persistent, the form accompanied with croupous casts of the bowel, and masses of thickened mucus, at times passed with much abdominal pain, at others the only constituent of the stools and passed almost without special sensation. With this condition, which may follow acute attacks but quite commonly occurs independently of all ascertainable cause, there are constantly symptoms of nervous debility, headache, complete loss of mental and muscular energy, anemia, and trophoneuroses which suggest that the malady is less a bowel disorder than a nervous functional disease. This is apparently supported by its occurrence after acute diseases with profound influence on the nervous system, as epidemic influenza. It is very apt to recur after apparent improvement, and mental worry will often bring it on quite suddenly after a previous attack. The ultimate prognosis is not good.

From acute proctitis the neighboring areolar tissue in the ischio-rectal pyramid is at times involved, with abscess formation and perforation into the rectum or through the skin.

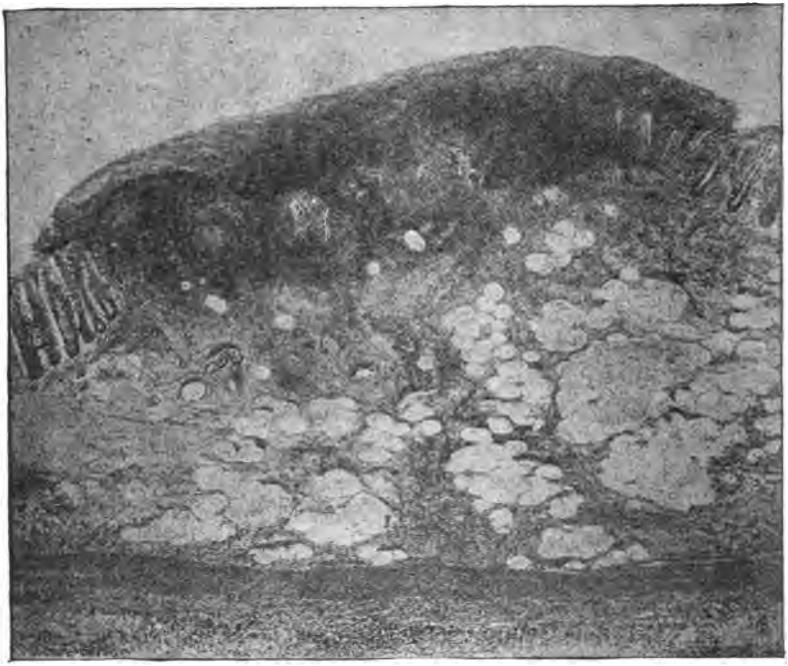
Enteritis Due to Specific Bacteria.

Dysentery.—This term is clinical rather than pathological and covers a number of lesions, which vary from each other, and yet show many transitions from one to the other. The specific organism varies also. It may be an ameba, as in tropical dysentery, invading the mucosa and submucosa, and causing severe ulcerative inflammation; at other times it is the bacillus coli communis which alone, or with b. pyocyaneus and cocci, is capable of producing extensive ulceration here and in other tissues. Still other cases have been described as due to a small bacillus (Ogati's) with rounded ends, actively motile, forming yellow-green colonies on gelatin plates. In other cases, chemical agents, especially mercury, produce ulcers and symptoms identical with dysentery, perhaps by destroying the mucous surface and permitting the bacteria to attack the tissue.

The mucous membrane in the milder cases may show little beyond reddening and edema, with slight increase of secretion, and in more severe only the solitary follicles may be the seat of ulceration. In the most pronounced cases the mucous surface is covered with a false membrane (hence the term diphtheritic enteritis as synonymous with dysentery), and under it the wall of the intestine is sloughing or gangrenous. All variations and combinations of these different grades are encountered. Hence dysentery is sometimes divided into catarrhal, ulcerative, croupous, diphtheritic, and gangrenous, according as the inflammation is superficial or deep, and the product simple increase of mucus or fibrin, serum and pus. In the specific disease of the pharynx known as diphtheria, similar inflammation of any part of the alimentary tract is very unusual, probably due to the destructive action of the gastric juice on bacteria which reach the stomach. Hence diphtheritic colitis refers rather to the coagulation of fibrin in and among the elements of the mucous surface, associated with necrosis of the same.

In the gross the colon may be covered here and there with a pseudo-membrane, easily stripped off or somewhat adherent, and under it there is a loss of substance constituting an ulcer; the ulcer may have begun in the follicles and remained there or spread; the edges of the folds of mucous membrane are necrotic; at times where the membrane has been removed there are long lines of ulceration; the mucous membrane is generally edematous, and in the early stages the hyperemia may have left hemorrhages in the tissue. The wall of the ulcer is irregular and edematous or necrotic, its base may be the muscular or even the serous layer. The entire colon may present these conditions or limited portions of it, and at times post-mortem changes make parts of it green while others are hyperemic or gray from the membrane; the whole makes a remarkably variegated appearance. When the ulcers are small and few it is common for them to heal and pigmented scars remain; even rather large losses of mucous tissue may be replaced in this way.

FIG 17.



NECROTIC COLITIS. (*Delafield & Prudden.*)

Circumscribed congestion and necrosis of glandular and connective tissue coats.

When the course of the disease has been chronic all varieties of inflammation and ulceration and partial healing may be found in the course of the colon. At times the ulcerating surfaces granulate for long periods and the stools are very purulent. Perforation of the intestine is not common even in severe dysentery. With such profuse ulceration over such a large surface supplied by the portal vein, purulent thrombi in the veins of the part often occur, pylephlebitis and secondary abscesses of the liver; this is true especially of dysentery due to amebæ, and pulmonary abscess may form in the same way. The ameba may be recovered from the stools or found in the tissue of the intestinal wall and in the secondary abscesses. It is large (20–30 μ in diameter) and presents a nucleus, vacuoles and inclusions of foreign matter, red cells, pigment, etc. On a warm stage it puts out pseudopods, at first limited to the ectosarc, then filled with the granular protoplasm; encysted forms occur. These organisms are probably swallowed in drinking water.

Asiatic Cholera.—The specific cause of this disease is the cholera vibrio and its characteristic lesions are produced in the small intestine, although the entire tract may be involved. Post-mortem examination, after death in the stage of asphyxia, demonstrates on the outer or serous surface of the intestines a peculiar mucilaginous film, perhaps more evident to touch than sight; the mucous surface is bright red or bluish in color with many small submucous hemorrhages.

The contents of the intestine may be copious, thin and watery, and contain small bits of desquamated epithelium, making the so-called rice-water discharge characteristic of the disease. Blood may be mixed with this fluid, rarely a coloring of bile. The rice water fluid is alkaline, has a peculiar stale odor, and contains sodium chloride, carbonate of ammonia and but little albumin (total solid matter from 1 to 2 per cent.).

The mucous membrane is specially swollen in lymphoid follicles, both solitary and agminate, when death occurs early (fourth day). The colon may be slightly hyperemic and edematous, filled with rice-water fluid from the small intestine; at times early in the disease pseudo-membrane forms and after the algid stage a secondary fibrinous enteritis occurs both in small and large intestine. The stomach may show punctate hemorrhages. The mesenteric nodes are somewhat swollen, firm and bluish or red. The microscopic picture is that of coagulation necrosis and desquamation of the epithelium, confined to the tips of the villi or occupying the entire thickness of the mucosa. In later stages of the disease the intestine may show but little of the rice-water material, bile is usually present, Peyer's patches are pigmented and diphtheritic membranes are found, in the lower part of the ileum especially.

The general appearance of the cadaver is distinguished by tonic convulsion of the flexor muscles, the arms and legs drawn up and the muscles hard, the hands partly clenched, the face sunken, nose and chin prominent, surface generally cyanotic or only the extremities. Hyperemia of the pia, lobular pneumonia, hyperemia and cloudy swelling in the kidneys, and shrunken liver and spleen are the common associated conditions. The vibrio may be obtained in pure culture from the intestinal mucus and the rice-water contents, if death has occurred early sometimes from the gall-bladder; in later stages it may be impossible to find (seldom after the twelfth day).

Typhoid Fever.—The characteristic lesion of this disease is found in the lymphoid tissue of the small intestine, especially in the agminate follicles (Peyer's patches or placques), just above the ileo-cecal valve (from 0.5 to 3 meters); but occasionally typhoid ulcers occur in the stomach or even the esophagus, more often in the follicles of the colon. In other cases the typhoid infection occurs through the blood or the bile capillaries and the intestinal lesions are not found.

Corresponding to the clinical stages of the disease, divided into weeks, the pathological lesions are so constant that it is fairly safe to estimate the length of the disease from the gross appearance of the intestine. In the first stage the mucous membrane of the lower ileum is actively hyperemic and edematous, the lymphoid follicles moderately swollen, the nearest mesenteric nodes large and very congested. In the second stage the lymphoid structures swell more and more, the general hyperemia may disappear and only the tissue about the glands be red. The Peyer's placques are prominent above the mucous surface on the side opposite the mesenteric attachment, and, as neighboring patches may join, the total length of such a swollen area may be enormous; the lumen of the gut may be lessened by the edema. The surface of the patch is smooth or foveolated, the edge usually sharply defined from the mucous membrane about. The color of these swollen lymph structures is at first translucent gray, then yellowish from fatty degeneration, afterward dull gray or yellowish as necrosis begins.

Under the microscope the entire thickness of the wall is infiltrated with small round cells and very hyperemic, in the follicular tissue there are also large lymphoid cells with round vesicular nuclei which do not take stains very well; between these the reticulum is at first preserved, afterward it is lost in necrosis; commonly the round-celled infiltration follows the large-celled.

In favorable cases the process ends here with disappearance of the edematous swelling, with granular and fatty degeneration and resorption of the infiltrating cells. In other cases the edema persists, even increases, and necrotic changes occur in the cellular infiltrate, leading to the formation of a slough which corresponds to the placque in outline, and, when this is cast off, an ulcer is left. Its edges are undermined, its floor is smooth, its main axis is parallel with that of the intestine, it lies opposite the mesenteric attachment and is usually confined to the patch; on the corresponding serous surface there is no fibrinous deposit and perforation is common—all these characters serve to distinguish the typhoid from the tubercular ulcer.

The ulcerative process may be accompanied with bleeding into the canal, if early from the hyperemia of the mucous membrane and hence not serious, if late as in the third week, from ulceration of a vessel in the floor of the lesion and possibly fatal. Perforation may occur as early as the end of the second week, during the third, or later, and may lead to general peritonitis or in some cases to a localized inflammation. Occurring without perforation the peritonitis is due to the deep character of the cellular infiltration, great loss of substance when the slough separates, and inflammation of the intestinal aspect of the serous membrane carried through to the other. The mesenteric lymph ganglia usually correspond in their size to the degree of intestinal inflammation, hyperemia, edema, fatty degeneration, suppurative softening or resolution in the nodes usually following similar changes in the patches. When the ulcers heal both they and the node are apt to be pigmented and this may persist for long periods. The nodes first affected lie in the angle between lower end of ileum and ascending colon; later on those near the root of the mesentery are involved; peritonitis may follow from suppuration in them.

The spleen almost constantly becomes hyperemic and very large and soft; infarcts and abscess may occur and the latter set up peritonitis. The muscles of the trunk often show areas of waxy degeneration, especially the recti abdominales, sometimes with hemorrhage; the skin may contain petechial hemorrhages and be necrotic over prominent points; the heart may be the seat of endo- or myocarditis; the blood does not show marked leucocytosis; bronchitis and lobular pneumonia are frequent complications; capillary apoplexies in the cerebral cortex occur; parenchymatous degeneration of the liver is frequent and the gall-bladder contains the typhoid bacilli; the kidney is the seat of degeneration or inflammation, the latter hemorrhagic in some cases, and inflammation and abscess of the parotid gland is found in others; periostitis and osteomyelitis are possible. After the patient is apparently convalescent pulmonary tuberculosis develops in quite a large proportion of the cases.

The typhoid bacilli may be recovered from the affected follicles, the mesenteric nodes, the spleen and the gall-bladder in almost every case; at times they remain for months in the intestine and appear also in the bladder and are voided in the urine; consequently promiscuous urination by typhoid convalescents may be a source of danger to the users of water thus contaminated.

A clinical study of the blood for diagnosis of typhoid fever has been lately introduced under the name of Widal's test or reaction. It depends upon the fact that serum from the blood of one ill with typhoid fever mixed with a recent culture will cause the typhoid bacilli to lose their motility and gather in groups, the whole called "clumping." Three drops of blood are taken from the well-washed aseptic finger tip or lobe of the ear, and each lies by itself on a sterile slide, passed through a flame and cooled just before use; this slide may be wrapped in cotton and transported for examination at the laboratory. Here one drop is mixed with a large drop of sterile water, to redissolve it; a drop from the summit of this is then mixed with six drops of fresh broth culture of the bacillus (not over twenty-four hours old) on a sterile slide; from this a small drop of mingled culture and blood is placed in the middle of a sterile cover-glass and this is inverted over a sterile hollow-ground slide and examined. The blood received as above or on paper should be diluted with twenty to fifty times its bulk to secure the best results; when received fluid, in a capillary tube, a drop may be taken with a fine pipette and nine drops of sterile water added; one drop of this mixture with one drop of broth culture nearly equals 1:20 dilution. A positive reaction is obtained when all the bacilli present gather in one or two masses or clumps, and cease their rapid movement, inside of twenty minutes, and the degree of clumping and time required should always be mentioned in reporting upon the reaction, as also the dilution employed. Sources of error make this test of value, but not entirely reliable. Obtained within forty-eight hours after the onset of a disease the treatment should be as for typhoid until the clinical symptoms demonstrate that the disease is not present.

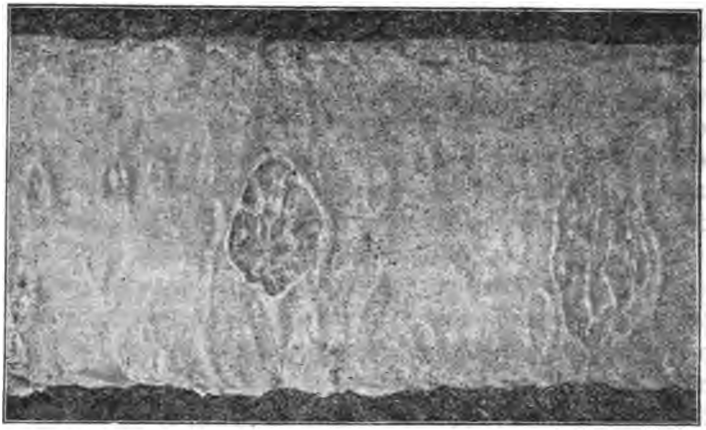
Anthrax Infection of the Intestine, Mycosis Intestinalis.—Certain trades which involve handling products of animals liable to anthrax, as wool, hair and bristles, hides and sausage "cases," involve the danger of swallowing dust containing anthrax bacilli or their spores. Infection of the intestine attacks especially the small and upper part of the large, and appears as areas of a few mm. in diameter up to several cm., on the mucosa of the attached side, prominent above the general surface, brownish-red or ulcerated and yellowish-green, here and there necrotic and of a darker green; from this site they may spread to encircle the intestine. On section marked edema of all the layers is found, the center is purulent and about it is a ring of hemorrhage. The serous coat may be hyperemic over such a place, or generally the rest of the mucosa is often red and swollen. The mesentery and its nodes are congested and swollen and the peritoneal cavity contains a small amount of serum, at times blood-stained. In some cases the bacilli are found especially in the summits of the valvulæ conniventes, in the gland lumina and the lymphatic channels, or deeper in the submucosa, in the lymph nodes and various organs. The brain and organs where they lodge may show the effects of the bacilli and contain vast numbers in the capillaries. The commonest source of infection is eating with unwashed hands, by which spores are swallowed with the food; bacilli, apart from spores, do not withstand the gastric juice.

Infectious Granulomata.—By far the commonest lesion of this variety is *tuberculosis* of the intestine. This may be primary from bacilli swallowed with food, as in milk from tuberculous cows, and hence most often observed in infants. The lymphoid tissues of the lower part of the ileum are especially liable to be affected, caseous and ulcerating foci forming in them and converting part or all of a Peyer's patch into a sluggish ulcer. Secondary infection is common in later stages of pulmonary tuberculosis with copious sputa, doubtless the swallowed bacilli are taken up by phagocytes and deposited in the lymphoid tissue.

If the solitary follicles are the seat of the ulcer it appears as a small round loss of tissue with thickened edges and a purulent base. Larger ulcers occupy the agminate areas and at times several become confluent.

As the process follows the lymph channels the inner side of the intestine is girdled by the ulcer, its general outline is oval, it lies with its long axis at right angles to the main intestinal axis unless produced in a placque by junction of several small ones, its edges are thickened and firm because of the fibrous hyperplasia and not undermined, the base is uneven and covered with pus and necrotic tissue, and on the corresponding serous surface there is an exudation of fibrin together with little lines and series of tubercles in ring form about the intestine. The ulcer may perforate, but the fibrinous process makes adhesions between neighboring coils or other parts and prevents the escape of fecal matter.

FIG. 18.



TUBERCULAR ULCERS OF THE INTESTINE. (*Stengel.*)

Ulcers oval, with long axis transverse, edges thickened and not undermined, bases rough.

Such large ulcers are not confined to the ileum; they are found as large and in numbers in the colon, and, when partly healed, firm scars, often deeply pigmented, occur alongside of more recent ulceration. These scars do not narrow the lumen of the gut perceptibly, as do syphilitic processes. The mesenteric lymph nodes are regularly involved and relatively seldom there is general tubercular peritonitis.

Syphilis of the intestine is rare, but most common in the rectum in adults; as this is the only portion of the tract exposed to direct infection it is the only site of intestinal chancre. The chronic ulcer developed from gumma of the wall is accompanied by round-celled infiltration and great thickening of the whole wall, papillary projections on the mucous membrane, ulceration and the formation of tough scars which contract and stenose the canal. In the new-born congenital syphilis may affect the duodenum and ileum, the lymphoid tissues being specially attacked.

Actinomyces rarely involves the cecum and produces the usual infiltration and ulceration. Enteromycosis is the name given to intestinal lesions produced by the ingestion of decayed fish, meat and other proteid food, accompanied by catarrhal or fibrinous inflammation and ulceration, profuse diarrhea and constitutional depression; the entire picture is rather that of toxin-poisoning than simple enteritis by any specific bacterium.

Tumors.—Benign new growths may develop from any of the structures of the intestinal wall; myoma, fibroma and glandular polypoid forms, at times combined with lipoma, are the commonest. A form of congenital anomaly occasioned by the constriction of a part of the gut is known as enterocystoma; it usually occurs as a small section of the intestine, atresic at each end and dilated with fluid, by the side of a normally functionating intestine.

Primary sarcoma is unusual, most often round-celled and at times congenital.

Carcinoma shows apparent preference for certain parts of the intestine, as the papilla in the duodenum, the ileocecal valve and its neighborhood, the line of junction between the sigmoid and the rectal portions of the canal. At the anal orifice the tumor may be an epithelioma, elsewhere it starts in the gland follicles (as Lieberkühn's) and soon surrounds the lumen while infiltrating the entire wall. Ulceration of the mucous surface is common, colloid and other degenerations occur oftener in the richly cellular forms, stenosis of the intestine is most marked in the scirrhous. Metastases in the mesenteric nodes and the liver are usually found. (See Pt. I., p. 166.)

Parasites.—Among the lower forms amebæ have already been mentioned as a cause of severe dysentery. Balantidium coli, cercomonas and trichomonas are also met with, associated with diarrhea, the cause of it or accidentally present; a better demonstrated relation between infusoria and inflammation occurs with the megastoma entericum. Worms are the most frequent intestinal parasites, and by their presence both local inflammatory and general nutritive and nervous effects are produced. Among the tape-worms tenia solium, mediocanellata and bothriocephalus occur most commonly in men; in Abyssinia a tenia nana is found in almost every person above a certain age and locally considered rather beneficial than otherwise. Nematodes, as round worms, may cause trouble by their great number, running into the hundreds, or by wandering into various lumina, or perforating the wall; for the latter result there is usually an ulcer or other defect necessary. Oxyuris causes chronic inflammation of the rectum in both sexes and wanders into the vagina in females. Trichinæ develop in the gut but do their worst damage in the course of their wandering into distant voluntary muscles. Anchylostoma may cause profound anemia and this is observed in certain countries (Italy) more than in others. (Uncinariasis.)

Foreign bodies in the intestine may be of any kind which can be swallowed and passed into the canal; or they develop as concretions about some nucleus, as a seed, by successive deposits of lime and magnesium salts; mixed with these are mucus and bile pigment in varying amounts so that their color is yellowish-white, brown or greenish. These *enteroliths* in lower animals reach a large size (as in ruminants); in man they are commonly small, ovoid, stratified, at times multiple, and of little importance unless wedged into the appendix. Sharp foreign bodies, as needles swallowed by the hysterical, often leave the intestinal tract and wander through the tissues, appearing after long periods under the skin of the thorax or limbs.

The fecal contents may be abnormally hard, forming scybalous masses, from long retention and consequent absorption of their fluid part; or abnormally soft and fluid from the addition of serous fluid, as after hydragogue purgatives and exudative inflammation. The gas normally present may increase to such an extent as to push the diaphragm upward and hinder respiration; this occurs suddenly in certain nervous states and slowly in typhoid and peritonitis. It is called meteorism or tympanites. Connected with it is often a paralysis of the muscular fibers of the intestine and cessation of peristalsis; stenosis of any part leads to much dilatation above it.

CHAPTER V.

ALIMENTARY GLANDS.

The Salivary Glands.

INFLAMMATION of the parotid gland (*parotitis* or mumps) occurs as an infectious disease, characterized by rapid swelling, which may pass to the tissue in the neighborhood and also to the other salivary glands. The inflammation is exudative and chiefly serous in mild cases; when severe, suppuration and abscess formation may follow. Edema may occur in the floor of the mouth when the lingual glands are involved. In adults so-called metastatic inflammation of the testicle and the ovary complicates some cases, and this occurs with the first inflammation or after it disappears.

As a secondary inflammation, parotitis is seen with several general infections, as typhoid and scarlet fevers, pyemia and tuberculosis, and congenital syphilis. The course of the inflammation may be from the larger ducts to the acini, after the former are plugged with exudate; when the tissues about the gland are also involved the swelling and pain, because so much of the gland is confined behind the angle of the jaw, become extreme. Such an acute inflammation may disappear and leave no trace, or pass into a chronic fibrosis (induration), or a suppurative stage which forms small abscesses throughout the gland and larger ones by their confluence. Gangrene of the part is occasionally observed in pyemia. At other times the limits of the gland are passed and the suppuration follows the fascia in the neck, or causes otitis or necrosis of the bones adjacent. The abscess may empty into the mouth or externally and fistulæ may remain. In some of these cases the infection is brought by way of the blood, in others the infection travels up the duct from the mouth.

The submaxillary gland may be involved at the same time with the parotid, or, in very unusual cases, by itself. An extremely severe form of independent inflammation of the submaxillary is known as *angina Ludovici*, and begins as a swelling in the gland, rapid suppuration and extension of the process to the entire suprahyoid region; in later stages it may also travel backward to the larynx and downward into the anterior mediastinum. Abscesses form which break into the mouth or externally, necrosis and gangrene are often associated, and death occurs, in about 50 per cent. of the cases, from edema of the glottis, pneumonia or septic intoxication. This form of angina may start in the submaxillary connective tissue, and perhaps the infection gains entrance through carious teeth, or, as in scarlet fever, it begins in the gland itself.

The sublingual gland may be inflamed by extension from similar processes on the tongue, seldom otherwise.

Hypertrophy of all the salivary glands may follow acute inflammation when the increase in size is chiefly interstitial, or, as in gum and tobacco chewers, it may result from functional activity long kept up beyond alimentary needs.

Tumors of the salivary glands are most often found in the parotid and primary forms are unusual in all. Benign tumors may be fibroma, lipoma, or chondroma, and mixed forms of these occur frequently with sarcoma of the part. Among the forms of sarcoma fibro- and melano- occur; adeno-sarcoma and epithelioma are sometimes found. Primary carcinoma is rare. Commonest among parotid tumors are the mixed forms which develop in children, of slow growth, without metastases (usually), and containing new cartilage, fibrous and sarcomatous tissue. They reach a large size and mechanically push their way into the pharynx and mouth. They may be explained as fetal inclusions and bear a relation to the outer end of the first branchial cleft.

The Salivary Ducts.—Fistulæ remain after abscess opening externally or elsewhere, or after operations which involve incision of the duct. Foreign bodies may make their way into the duct and cause inflammation, and concretions sometimes form here by deposit of mineral salts about a nucleus; these are called sialoliths, and are found both in the main ducts and deeper in. If such a foreign body or concretion causes stenosis of the duct the secretion collects behind it and dilates the part, with pressure atrophy in the acini. Such a retention cyst is called ranula and is most common in Wharton's duct. Another form of ranula begins in a mucous pouch which is occasionally present near the frenum linguæ.

The Pancreas.

Malformations.—The commonest in viable children are accessory lobes of the main gland, accessory glands sometimes with ducts of their own, formation of pancreatic tissue in the intestinal wall (duodenum), and variations in the main duct; this may be double or may open into the common bile-duct or the stomach or in unusual parts of the duodenum. In about sixteen per cent. of all cases there are two pancreatic ducts, of Wirsung and of Santorini, opening separately into the duodenum.

Disorders of Circulation.—Functionally hyperemia occurs after a meal, relative anemia after digestion ceases, actual anemia in starvation and cachexia. Hemorrhage in and about the pancreas may result from contusion over the site of the gland or from disorganization of the blood. Suppuration may open large vessels and cause fatal hemorrhage, as is sometimes seen in advanced fat necrosis involving the gland.

Inflammation.—An acute form with great swelling, hyperemia, and suppuration later, is known as *pancreatitis purulenta.* The pus appears in small points through the organ, which may become confluent, or the interstitial tissue is uniformly infiltrated with purulent exudate; such an inflammation may be primary or extend from other organs, and when gastric ulcers perforate the posterior wall of the stomach this form with gangrene sometimes results. Embolic abscesses and a purulent inflammation of the peripancreatic tissue also are observed. Occasionally these cases develop with great suddenness, perhaps most often in men of obese build, and simulate acute intestinal obstruction.

Chronic interstitial pancreatitis presents a stage of infiltration, when the gland is larger than normal and somewhat firmer in consistence, followed by a stage of contraction of the fibrous tissue and a noticeably atrophic organ. Alcoholism, syphilis, and ascending inflammation after gall-stones lodge in the ampulla all seem to be productive of this cirrhotic hardening of the pancreas. The new tissue may begin about the blood-vessels, and as it increases, especially when contracting, the glandular elements degenerate and disappear.

Tuberculosis is found in the pancreas as part of a general infection as almost microscopic miliary granules in the glandular tissue, or aggregated in larger masses which may soften in the center.

Syphilis of the new-born very seldom affects the pancreas, but gummata have been described both in these cases and in acquired syphilis. The fibrosis which occurs in adult life has been mentioned; this at times accompanies similar changes in the liver and from syphilis as one cause. At times congenital syphilis includes such a hardening of the gland.

Tumors.—In the obese, as chronic beer drinkers, the pancreas may share in the general lipomatosis of the abdominal organs, usually combined with atrophy or replacement of the functionating epithelium; in late stages but little of the original structure may remain.

Sarcoma is one of the least usual tumors of the pancreas; it may be round-celled or adeno-sarcoma. Secondary sarcomata occur with general spread of the neoplasm.

Carcinoma on the other hand is not uncommon, after similar disease of the gall-bladder, stomach and intestine; primary carcinoma may occur either in the head of the organ or in other parts, and the hard fibrous forms are most common. If it starts in the wall of the duct the cancer is usually scirrhous, if from the acini it may be alveolar, containing spaces clothed with cubic and polymorphous epithelium in several layers. From the pancreas metastases into the liver, the gall-bladder and the duodenum usually occur. If the tumor is cystic it may be due to retention of secretion and dilatation of the duct, or to degenerative softening within the neoplasm.

Degeneration.—Typhoid and yellow fevers, variola and diphtheria, and other infectious diseases are frequently accompanied by granular and fatty degeneration as in the other parenchymatous organs. In the gross the pancreas may be larger than normal, soft and of a dull grayish color, and under the microscope the cells are filled with fine granules or fat. Chronic phosphorus-poisoning causes marked fatty degeneration.

Necrosis affects scattered areas of the organ or large sections, and the causes in the two cases may differ. When a large portion is necrotic it may be from inflammation near by which has extended to invade the gland, as with gastric ulcer. In other cases a peripancreatitis leads to partial or entire death of the organ. In others a post-mortem self-digestion simulates extensive necrosis.

When the flow of secretion from the gland is interfered with by contraction or obliteration of the duct of Wirsung (and of Santorini when present), the fluid backs up and dilates the containing channels. If the stenosis is temporary no effect may be produced in the gland, but after a time the epithelial cells lining the ducts and in the acini may degenerate and be cast off and thus the digestive ferments are able to attack the tissue. The steapsin causes a peculiar form of destruction of the fat, called *fat necrosis*, which appears as small dull gray spots scattered through the fatty tissue; at first these may be found only between the lobes of the gland, and are far more frequent than usually stated; if the pancreas were carefully examined at every autopsy their occurrence would be more often noted.

Later the omental fat is attacked and shows the same whitish or gray foci. These are one or two mm. in diameter, clearly marked off from the shiny peritoneum, sometimes gritty under gentle palpation from contained lime salts of fatty acids. The steapsin splits the fat into glycerin and a fatty acid; the latter may unite with alkaline bodies in the tissue. More extensive necrosis, the cause persisting, at times accompanied with purulent exudate, destroys the gland or opens large vessels (veins) and gives rise to bleeding which may be rapidly fatal. The blood may escape into the peritoneal cavity, or it may dissect behind the peritoneum along the sheaths of the psoas muscles into the pelvis, spongy clots and fluid blood filling the artificial cavity. All that remains of the pancreas may be a small portion of the head in the curve of the duodenum and a bit of the tail in the hilum of the spleen; the portion between is converted into a mass of necrotic tissue and clot. From the nature of the process there are no clinical symptoms for a long time and the course of the disease can not be estimated. Microscopically the small areas at the beginning show hyaline places which were occupied by fat cells, fairly preserved cells enclosing fat crystals, calcium compounds with fatty acids which take hematoxylin strongly, and a zone of hemorrhage about all. In subacute cases there may be areas of cystic softening in the omental or other fatty tissue, containing thin puriform fluid and necrotic shreds.

Amyloid degeneration occurs as spots and streaks of dull gray color and firm feel on the cut surface of the organ; it is found with other amyloid degenerations, seldom alone, and does not reach so marked a degree as in other organs.

Pigmentation occurs in old age with atrophy and in general deposit in other organs, from similar causes. There is seldom any iron in the pigment.

Atrophy may be simple as in cachexia and senility, or associated with replacement of gland by fatty tissue.

Some of these forms of degeneration may be observed after death from diabetes, and experimental removal of the gland in dogs causes glycosuria in most cases which is rapidly fatal. It is supposed that the islands of Langerhans, which in the human organ lie almost exclusively in and about the head of the gland, have a peculiar function in regulating the metabolism of sugar, and that when degenerative changes in the pancreas are accompanied by glycosuria it is because that section is involved where these cell accumulations are found, and that rather extensive pancreatic lesions limited to the rest of the gland may explain the absence of diabetes even with marked disease in this organ. The well-established fact that diabetes is often accompanied by lesions in and about the fourth ventricle prevents our ascribing all cases to pancreatic disease. The pancreatic lesion is not always of one kind, even in the pancreatic form of the diabetes, but certain ones have been found both in obese and in cachectic patients.

The duct of the pancreas presents about the same anomalies and diseases, concretions and stenoses, as do those of the parotid and other salivary glands; even a form of pancreatic ranula has been described. Catarrhal inflammation of the crypts of the gland may result in multiple cystic tumors all through the organ, very small and with clear or purulent contents; the latter may thicken and become caseous.

The Liver.

Malformations.—The organ may be entirely lacking in extreme general malformation; variations in the size and number of the lobes is common, accessory masses of hepatic tissue may be found in the neighborhood, usually connected with the main organ by pedicles. Complete situs transversus involves transposition of the liver; other changes in location occur in fissures of the diaphragm, hernia, and as an acquired form from external pressure of belts and corsets. The line of greatest pressure usually runs obliquely across the right lobe on the anterior surface and at or below the middle of the vertical height, making a groove where the capsule is much thickened and the balance of the lobe below swollen and indurated; the gall-bladder may be distended. Another form of groove occurs on the superior aspect of the right lobe under the dome of the diaphragm and in some cases may be explained as the result of long-continued dyspnea, irregular and convulsive action of the muscular diaphragm crowding portions of the liver tissue together and making acute-angled furrows between them; these may be as many as five or six, each bounded by round prominent ridges of hepatic tissues, running from before backward and below upward. In other cases these grooves are noted in young children who can not possibly have had dyspnea of such a degree as to have caused them. In the former case the furrows do not lodge specially strong bands of muscle, in the congenital form they usually do and hence are broader and shallower. The gall-bladder is sometimes lacking; the common duct may open into the stomach. Among other acquired changes in the location of the liver may be mentioned looseness of the ligaments, which permits the organ to fall, downward dislocation from fluid in the right pleura, misplacement connected with kyphosis and other spinal curvatures, and the rotation upward about a transverse axis caused by ascites, pregnancy and tumors in the abdomen.

Disorders of Circulation.—The liver resembles the lung in developing as a bud from the alimentary tract and in having two circulations, one for its stroma, relatively unimportant, and one for its function of far greater capacity and importance, with but little communication between them, and lymphatics to correspond. The portal system is the largest element in the hepatic circulation, contains blood which has already passed the capillaries of the other abdominal organs, and hence is characterized by slow flow of its blood under but little pressure; to this the rapid increase of its total capacity within the organ, due to the division of the main trunk into many smaller ones and these into thousands of large capillaries, of course contributes. Consequently very slight degrees of hindrance to the exit of blood by the hepatic vein causes marked general passive hyperemia of the liver. Pigment, parasites and bacteria derived from the portal territory are almost constantly deposited in the organ.

Anemia plays but little part in the pathology of the liver; it may occur locally from pressure of tumors or generally after loss of blood. The gross appearance depends in part on the lack of blood but even more on the proportion of contained fat, bile and pigment; being pale with a tint of yellow, green or brown, with increased firmness and diminished size; the gall-bladder may be empty or contain a little thickened bile.

Active hyperemia occurs locally about inflammatory areas, tumors and wounds; in certain infectious diseases as typhoid and malarial fevers and hepatitis it may be general. The organ is swollen, of bright or darker red, soft and friable, and on section blood flows freely; such a liver begins to decompose soon after death in favoring circumstances and the large veins may contain froth from the presence of bac. aërogenes capsulatus.

Passive Congestion.—The underlying cause in the great majority of cases is interference with the blood flow through the hepatic vein. This may be direct from pressure upon it or the vena cava inferior, as by right-sided pleurisy with effusion which pushes the heart to the left and drags on the vena cava; or it may be indirect and due to mechanical obstacles to the emptying of the right cavities of the heart, as in emphysema, pneumonia, valvular disease and aneurysm. The common result of such mechanical conditions is dilatation of the central vein in each acinus, then of the capillaries immediately connected with it; owing to the steady pressure thus exerted upon the liver cells they atrophy, at first about the central vein, in long-standing cases even to the periphery.

In the gross such a liver shows dark red points and figures, like letters and portions of letters, due to the unequal division of the central veins by the knife. By slant illumination these dilated vessels are seen to occupy slight depressions due to the atrophy of adjacent cells; further out there is a zone of brownish color owing to the presence of atrophic liver cells containing pigment; along the periphery there is a lighter zone of fat infiltration and bile staining. The total effect is that of dark red marks on a lighter ground and in severe degrees the name "nutmeg liver" is used from a fancied resemblance to the cut surface of a nutmeg. According as the passive congestion is marked the size of the liver is increased; with atrophy it becomes smaller and firmer, hence the appearances vary with the stage of the condition.

Infarct of the liver from closure of arterial branches is practically unknown, owing to the rich anastomoses between these vessels. Even large thromboses of the hepatic vein may remain without pronounced effects; but small scattered areas of necrosis may be due to embolism in the hepatic capillaries of the portal vein. Similar focal necroses occur in puerperal fever, septicemia from lesions in the portal territory, and arsenic-poisoning. These may be anemic or hemorrhagic, the former appearing as circumscribed points of pale yellow color throughout the organ with hyaline thrombi in adjacent vessels (capillaries) in many cases; the other form is mixed in color, partly red, from extravasated blood, and partly yellow or paler, from the coagulation necrosis of the liver cells. Such focal necrosis is often most prominent in puerperal diseases.

Edema of the liver follows marked and prolonged disturbances in its circulation and is a part of inflammatory processes; microscopically the cells may present vacuoles and the lymph spaces show distention by lymph as elsewhere.

Occasionally, a liver presents under the capsule and through the parenchyma a number of brick or brownish-red irregular markings and spots due to extravasation of blood and local alteration of the hemoglobin; it is usually the sequel of a chronic process and is to be referred to previous not determinable periods. Other instances of pigmentation are found after chronic malaria, blood disorders with hemolysis, and anthracosis.

Injuries.—Wounds of the liver which permit bleeding may be rapidly fatal, though there are instances where apparently severe cuts and stabs were healed. By forcible contusions the organ may be ruptured and the bleeding is usually extreme, the blood collecting under the capsule if this is not torn or being found free in the abdomen. Such a hematoma is not uncommon in difficult births, especially breech presentations with instrumental delivery. The injury may be a small subcapsular tear, often on the anterior superior surface, or the capsule may be torn and the liver tissue present a ragged single crush or many small wounds; the bleeding comes especially from the thin-walled veins and is usually fatal. Bits of the liver tissue may be forced into the hepatic vein and cause embolism of the lungs similar to the post-mortem condition of the like nature.

Inflammation.—Acute inflammation of the liver may occur with injuries, but most commonly it follows acute disease in the intestinal canal, as dysentery or suppurative processes, and appears as a single large abscess or as many small ones. The usual seat of the abscess is the right lobe and its size may be very large. The first step is a necrosis of the liver cells, granular degeneration with loss of nuclei, with an infiltration of small round cells from the interlobular and intralobular capillaries; pus-cells come later, in the vessels and then free in the tissue, and with their increase a focus of suppuration is formed; by pressure on the liver cells this induces a degenerative change, and thus increases in size, rapidly or in a slow chronic manner. The pus is usually thick and more or less tinged with bile. The result may be rupture into the abdomen, or through the abdominal wall after adhesions form, or into the thorax, or formation of a capsule and desiccation of the pus with calcareous deposit.

Suppurative phlebitis of the portal vein (pylephlebitis) causes another variety of hepatic abscess, the organ then being filled with dilated channels containing pus with purulent reaction about them. Abscess at times occurs with parasites or from gastric ulcer, and in pyemia with suppurative foci at distant points; the pyogenic cocci often accumulate in the liver capillaries and cause multiple secondary abscesses. In any case the hepatic tissue about the collection of pus presents a zone of intense congestion or even hemorrhage, with beginning fibrous hyperplasia if chronic, and in the acute cases all the liver cells may be in a condition of granular or fatty degeneration.

The microörganisms found may be cocci, especially streptococci, bacillus coli, various forms from the intestine and amebæ in tropical dysentery; in chronic cases the pus may be apparently sterile.

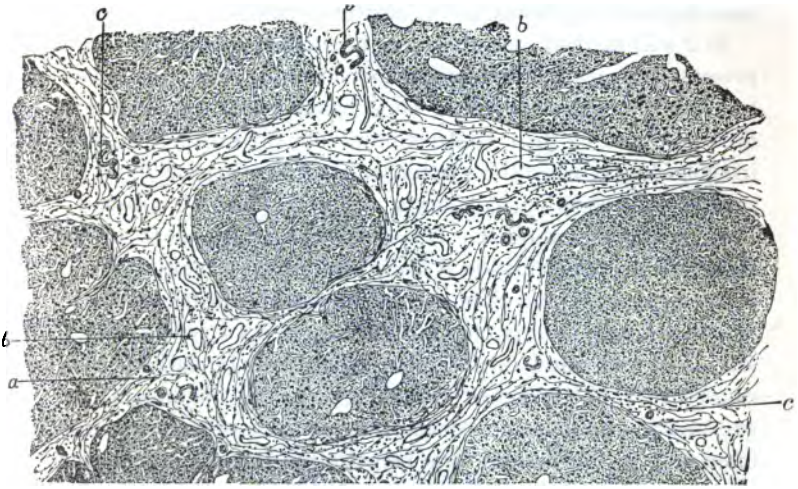
Chronic inflammation of the liver is productive and in slight degrees occurs as an induration with chronic stasis, but as a clinical entity it is described as cirrhosis of various forms; these are similar in histology but their gross appearances and clinical symptoms differ.

Cirrhosis.—The two chief forms recognized are the hypertrophic, and the atrophic.

Hypertrophic cirrhosis is also called hepatogenous, the irritant which causes the disease being supposed to arise in the liver, and biliary from icterus and hyperplasia of the bile-ducts. In the gross the organ is enlarged in all its dimensions, the sharp anterior edge is rounded and the weight may be as high as 4–4.5 kilos; on section it resists the knife, but less than in the atrophic form, and the cut surface is mottled with green, yellow and brown in varying proportions. Close inspection reveals the newly formed fibrous tissue along the line of the portal capillaries and the bile-ducts quite uniformly disseminated throughout, and by the contraction of this the little island of cells, more or less stained, becomes slightly prominent on the section; this arrangement has given the name "unilobular" as a synonym, since the acini are involved one at a time, as distinguished from the atrophic form where the fibrous tissue surrounds many lobules and hence "multilobular" is the term employed. Both the surface of the liver and the cut section in this form are smooth and there is no reason to suppose that a smooth hypertrophic liver ever becomes one of the nodulated atrophic form. The gall-bladder may be contracted but usually contains thin bile and the larger ducts are patent. The lymph nodes in the transverse fissure may be enlarged.

Microscopically the bile-ducts show two changes—they are increased in number and caliber, and many of them are plugged with pigment and cell detritus; about most of them there is an evident proliferation of fibrous tissue, which suggests that the lesion is essentially a pericholangitis. Many bacterial forms have been recovered from such livers and are probably the exciting cause of the lesion. The fibrous tissue may be traced far into the acini between the liver cells as is not often the case in the atrophic form. Here and there a hyperplasia of the liver cells may be found but in the main there is marked atrophy and pigmentation of them. In the late stages the bile-ducts show dila-

FIG. 19.



ATROPHIC CIRRHOSIS OF THE LIVER. (*Delafield & Prudden.*)
a, new formed connective tissue; *b*, dilated blood-vessels of the new tissue; *c*, gall-duct.

tation, hyperplasia of atypical lining epithelia, adenomatous grouping, and suggest transitional forms between cholangitic hyperplasia and primary carcinoma starting in the biliary epithelium.

Atrophic cirrhosis, known also as gin-drinker's liver, is sometimes described as hematogenous on the hypothesis that the causal irritation is brought to the liver through the blood, and in the majority of cases this is considered to be alcohol ingested with food and carried through the portal vein. Beverages with a large per cent. of alcohol taken into an empty stomach are specially liable to cause atrophic cirrhosis. It is supposed that the poisonous element in the alcohol is a volatile oily aromatic body known as furfural ($C_6H_4O_2$) which occurs as an impurity in almost all distilled liquors, hence the use of wine or beer does not commonly cause hepatic cirrhosis.

In a well-marked case the liver is smaller than normal and presents both marked induration and shrinking, the new connective tissue being disposed in large bands which run in various directions and by their contraction divide the organ into many irregular lobes; when these latter are small they project above the surface and resemble nail heads, hence the name "hob-nail" liver, sometimes applied. Owing to the arrangement of the fibrous bands the condition is sometimes called "multilobular," since many acini at a time are grouped and constricted by it. The liver tissue, sharply circumscribed, presents a yellow color if fatty, and with admixture of bile pigment may be darker. The capsule may be thickened or adherent to the diaphragm, the gall-bladder presents no constant appearance except that it is often empty. Adhesions form between the abdominal walls and contents and tend to relieve the portal stasis for a time.

Microscopically the formation of connective tissue may be traced in all its stages in the line of the portal subdivisions, in the youngest places appearing as a focus of round cells about the sheath of the veins with later development of spindle cells. These foci blend and the fibrous tissue becomes poor in cells and firmer, coincident with contraction. This causes compression of the portal capillaries and at the same time there is often an obliterating endophlebitis; the result is to offer an obstacle to the entrance of the portal blood with increasing stasis throughout the origins of the system. This explains many of the clinical symptoms, the ascites, gastric and intestinal catarrh and hemorrhage, piles, and distention of superficial abdominal veins (caput medusæ) to which latter effect ascitic distention adds. Jaundice however appears late if at all and the spleen is not swollen till toward the end; in the hypertrophic form jaundice appears early and is a marked symptom, the spleen is enlarged from the first and the gastric and intestinal hemorrhages seldom occur. In other words the portal circulation suffers especially in the atrophic form and the bile-ducts in the hypertrophic.

A resumé of these two forms will assist in distinguishing them:

	Cirrhosis.	
Synonyms.	Charcot's, Hypertrophic, Unilobular, Hepatogenous, Biliary,	Laennec's, Atrophic, Multilobular, Hematogenous, Hob-nail liver.
Jaundice.	Early and marked, bile often absent from feces.	Late and slight, bile usually present.
Ascites.	Late and unimportant.	May be early; often enormous.
Spleen.	Enlarged early and markedly.	Late and less.
Alimentary hemorrhage, piles.	Not common.	Common.
Liver.	Large, smooth, mottled, green.	Small, rough, pale or yellow.
New fibrous tissue.	In fine lines and strands between acini and cells, involving all parts equally.	In broad bands, making prominent islands in which the single acinus may appear nearly normal; distributed irregularly.

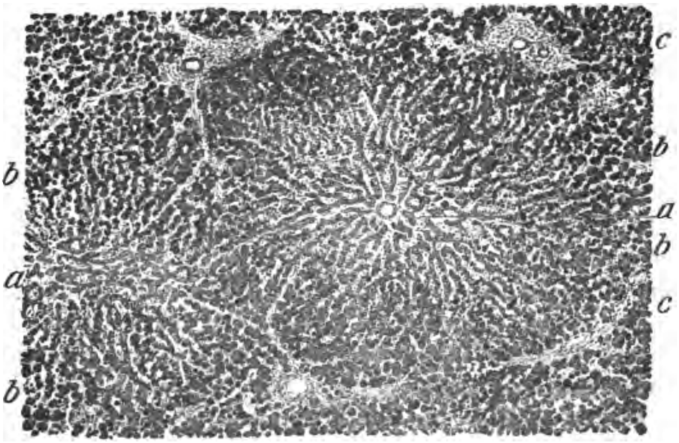
Other forms of cirrhosis have been described, a "paludic" in chronic malarial pigmentation of the organ, an "anthracotic" when pigment taken in through the lungs gains access to the liver and acts as an irritant, and as a complication of diabetes and with great thickening of the capsule (perihepatitis); the essential feature in all is the formation of new fibrous tissue in either of the two ways described above. When the main bile-ducts are compressed or obliterated the biliary stasis which results, with disorganization of the bile, causes minute peripheral foci of necrosis in the liver and a hyperemia about them; in these foci new fibrous tissue begins to develop and a form of cirrhosis may result which belongs to the "biliary" group, in which the irritant exerts its influence from the bile-capillaries. If pyogenic organisms gain an entrance from the gut before the chronic productive inflammation occurs the result is suppurative pylephlebitis and multiple hepatic abscesses. The liver in biliary cirrhosis of this variety differs from the hypertrophic form in being smaller, with less pigmentation and general icterus, and in shrinking with the progress of the disease. (See syphilis, p. 182.)

Degenerative Changes in the Liver.—*Simple atrophy* of the liver occurs with starvation and cachexia of many kinds, the organ being small, its glycogen lost (normally about 3 per cent.), its cells fatty or shrivelled. In a localized form such atrophy follows pressure if exerted for a long time and more generally it is a senile change. In the atrophic liver there is often an excess of pigment derived from the blood and containing iron.

Acute yellow atrophy is a rare disease accompanied by symptoms which suggest infection by bacteria, namely, high fever, jaundice, stupor and convulsions, hematemesis and bleeding from the bowel, the changes in the liver being rapid disappearance of the hepatic cells, softening and yellow hue of the entire organ. In the gross the separate acini can not be made out, the liver is smaller than normal, it is bright yellow or streaked with red or brown or uniformly of dark yellow brown hue, the capsule is wrinkled and the vessels contain a little thin fluid blood. When bile is present in the gall-bladder it also is very thin and pale. To this combination of pernicious clinical character and hepatic changes the name acute yellow atrophy is given, and its septic nature is evident from the association of the disease with puerperal and other severe infectious invasions. In other cases it is part of a rapidly fatal syphilis or follows extreme icterus, and it occurs also by itself without apparent cause. Many varieties of microörganisms have been found in the bile passage and the liver substances but none has been proved to be the cause. The above is the usual appearance in rapidly fatal cases but at times the liver exhibits areas of red or hemorrhagic appearance alternating with yellow portions, or the bulk of the organ may be of the former color with but small foci of yellow.

Microscopically the yellow parts contain swollen bile-stained cells filled with granules or fat drops or they are converted into detritus and bile crystals. If this material is absorbed the part decreases in size and all that remains are the blood-vessels, often impervious, mingled with grains of pigment ; these are the older and redder portions, in which also there may be remains of hemorrhages.

FIG. 20.



FATTY LIVER. (*Ziegler.*)

a, central portions of acini ; *b*, peripheral fatty zone ; the fat appears black ; *c*, interlobular connective tissue.

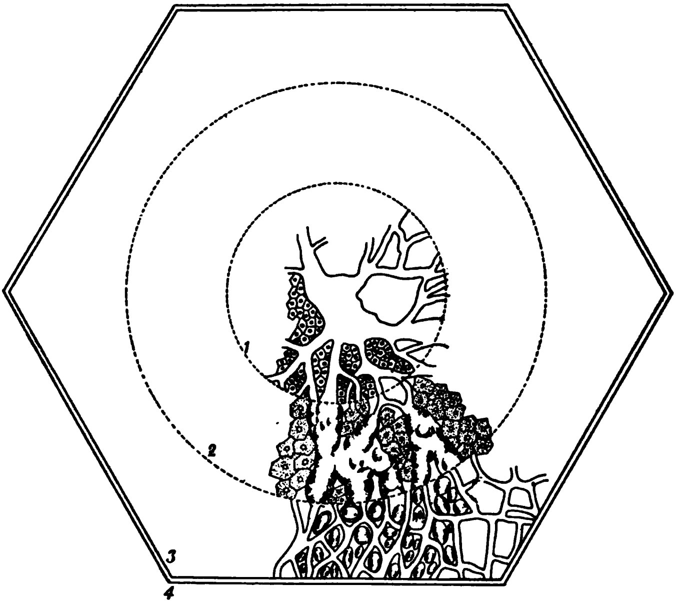
Fatty liver occurs in two forms, which in their most marked development may not be distinguished by the naked eye and yet are radically different in origin and importance.

Fatty Infiltration.—The fat normally present in the liver after a meal is a physiological fatty infiltration and appears most clearly in the peripheries of the acini; if the supply of oxygen is good and the function of the organ normal it is transformed and removed. When the supply of fat is relatively large or its combustion imperfect this fatty infiltration of the peripheries of the acini reaches a higher degree. Such a condition may coexist with general emaciation, as in the tuberculous who live on milk, cream, eggs, cod-liver oil, etc., but attains its most marked development in the case of the chronic beer drinker, where the weight of the liver may reach twice its normal (3–3.5 kilos). The organ is large, its edges are rounded, it is of a uniform yellow color, slight pressure leaves a dent in it, the knife becomes smeared with fat on cutting into it, and it may even float in water and burn freely when held in a flame. If not excessive the fat will be found in the outer zone of each acinus and on dissolving it out with ether the cell with its nucleus will be found; with dilated central veins the nutmeg liver is formed. In later stages all gross distinctions are lost in the uniform buttery mass and the cells have many of them suffered in nutrition, partly because the organ is anemic from the pressure exerted on the blood-vessels.

In *fatty degeneration* the liver is usually smaller than normal, yellowish, friable, and on microscopic examination the fat occurs in smaller droplets in the cells, which are also the seat of albuminous degeneration. The fat is found disseminated through the organ, not confined to the peripheries of acini, and other organs show similar degenerations, the whole being part of general diseases like pernicious anemia, variola, pyemia, yellow fever, etc. The distinction is easily made with teased preparations even without reference to other organs. Severe fatty degeneration occurs with phosphorus-poisoning.

Amyloid Degeneration.—As elsewhere this change follows long-continued cachexia, suppuration, tuberculosis, and is associated with similar degeneration of other organs. It has its seat in the walls of the vessels, the hepatic artery, its capillaries, the portal branches, but in the case of man does not attack the hepatic cell. When pronounced the liver is large, its borders are rounded, it is firm, inelastic, and on section of a pale and dull yellow or a brownish or grayish-red. The middle zones of

FIG. 21.



PATHOLOGICAL ZONES IN THE HEPATIC ACINUS.

Diagram of a lobule as a six-sided prism, cut transversely, entirely surrounded by fibrous tissue (this is not the case in the normal human liver). 1, circle of dilated central vein (passive congestion), stasis, and pressure atrophy; vein appears dark red and slightly depressed: 2, zone of amyloid degeneration, the vessels converted into swollen masses of amyloid, waxy in the gross; 3, zone of fat infiltration; appears light yellow in the gross; 4, line of connective tissue increase, pale and gray on fresh section. All these limits are exceeded in advanced lesions.

the acini are the first affected, and the hepatic cells undergo atrophic and degenerative changes from pressure combined with lessened nutrition. The walls of the vessels involved are converted into dull and swollen strands in which no lumen may be found.

Infectious Granulomata.—*Tuberculosis* of the liver is secondary to infection of other organs or part of a general miliary disease and appears either as miliary tubercles, sometimes almost invisible to the naked eye, or larger forms with a zone of inflammatory reaction, or old caseous foci. In the chronic forms, which often follow tubercular peritonitis, cirrhotic fibrosis is common.

Leprosy produces nodules in the liver, as in the spleen, which appear to start in the connective tissue of the organ and contain the bacilli, as do the capillaries of the part.

Syphilis is the most important of the granulomata found in the liver and occurs as disseminated cirrhotic inflammation or as localized gummata. In the cirrhotic form (hepar lobatum) the liver is variously distorted and divided into lobes by scar-like bands. These may nearly divide a portion of the organ from the rest and the irregular disposition of the new tissue distinguishes it from atrophic cirrhosis, of the alcoholic type. Early stages of the lesion are seldom encountered, but probably the process begins as a gummous inflammation about the branches of the portal vein and the main gall-ducts, with increased size of the organ and thickening of the capsule.

Gumma occurs as localized lesions from a few millimeters to several centimeters in diameter and also in combinations with the former lesion. The centers of the larger nodes may be caseous and about them, especially when recent, there is a zone of hyperemia. Microscopically giant cells occur, less freely than in tubercles, many eosinophile cells, necrotic liver cells, and spindle cells peripherally. In the older foci the necrotic process leaves nothing distinguishable but a fibrillary stroma filled with cell detritus.

Congenital syphilis affects the liver in diffuse forms and as gumma and also as miliary points throughout the liver or gathered in groups resembling tubercles, and in the majority of cases seems to begin in the portal capillaries and is associated with pulmonary syphilis. In an acute form with severe fatty degeneration the so-called acute yellow atrophy of the liver in the new-born has been described though it is really congenital syphilis of the organ.

Lymphoma of the liver occurs at times with acute diseases, such as typhoid and variola, and is regularly found in leukemias.

Tumors.—True hypertrophy of the liver is most unusual except as partial compensation of one part when another has been destroyed.

Primary tumors of all kinds are not common. The benign include fibroma and lipoma, and, more often, angioma of the cavernous form, which may reach a large size.

Sarcoma is at times primary in young children and several varieties have been described; secondary tumors of this kind occur with sarcoma of the kidney, testicle, bones and eye (often melanotic) and as in the case of secondary carcinoma are at times due to transfer of microscopic masses of neoplasm and embolism of portal capillaries.

Adenoma occurs in nodules of a reddish or grayish color or diffusely, or in combination of these two. When a cirrhosis coexists the gross appearance resembles that of carcinoma, and even microscopically there is difficulty in distinguishing them at times. A form still more uncommon is the *hypernephroma* which develops from portions of adrenal tissue included in the liver during fetal life. In site they usually correspond to the right adrenal and are apt to be encapsuled; they may be pure or enclose also hepatic cells and gall-ducts.

Primary *carcinoma* of the liver is rare. It occurs as one or more large tumors, or diffusely with increase in the size of the organ and fibrosis, and in an interlobular form which probably arises in the bile-ducts. Perhaps development on a cirrhotic basis is the most common, the gall-duct epithelium being stimulated to hyperplasia instead of degenerating, and leading to adenoma or carcinoma or combinations of these. The characteristic of carcinoma starting in this way is the irregular arrangement of its elements whereas in adenoma a stroma with glandular alveoli, clothed with atypical epithelia but still fairly regular in arrangement, is more common. Other forms appear to develop from the hepatic cells and present great polymorphism in their epithelioid elements. Fatty and other degenerations, biliary and melanin pigmentation are observed in these tumors. The liver tissue may be atrophic from the pressure, the tumor far exceeding the liver tissue in mass, or it may be hyperplastic in places and degenerated in others. Vast numbers of new-formed bile-ducts occur in some forms, especially where there has been a preceding cirrhosis and new growth of the ducts. Primary carcinoma is perhaps more common in the left lobe than the right, but rare in any case.

Secondary carcinoma follows similar disease of the stomach, pancreas, intestine, kidney and more distant parts. It is most often seen in the right lobe or in both when observed later in the disease, and may be a few large tumors or many small ones; it frequently corresponds in its distribution to the portal vessels and carcinomatous emboli may be found in them, as well as places where such emboli are invading surrounding tissue.

Cystic tumors in the liver are unusual and seldom of any great size; they start in the gall-ducts, perhaps also in lymph channels, and usually contain clear or yellowish fluid.

Parasites.—Ameba coli occurs in the liver in tropical dysentery both in the tissue and in abscesses. The psorospermiæ of lower animals (rodents) have been found. Pentastomum denticulatum, the larval form of the dog's p. tenioides, occurring in small cysts, distomum hematobium in the portal vein, distomum lanceolatum (in China and India), and distomum hepaticum of ruminants (sheep), have all been described as occasional inmates of the liver tissues. The most important parasite is perhaps the echinococcus which occurs in the three forms e. scolicipariens, being the larval form of the dog's tenia echinococcus, the e. hydatidosus, and e. multilocularis. The results of such cystic growths, which may attain a large size, are partly those of compression, causing icterus and ascites, and partly those due to rupture, into the peritoneum, the vena cava with general dissemination, and elsewhere. If pyogenic bacteria are present, abscess formation complicates the disease ; in other cases the fluid may be absorbed and the site remain as an irregular scar.

Gall-bladder and Ducts.

Changes in Caliber.—Stenosis of the main gall-ducts occurs usually from outside pressure, as by the contractions of inflammatory adhesions, enlarged lymph nodes, various tumors of the liver and adjacent organs. If the cystic duct is closed the other ducts are but little dilated but the gall-bladder may become much distended by mucous and serous fluid, either bile-stained or clear ; this is called dropsy of the gall-bladder. When the hepatic duct is compressed the entire system above the obstacle dilates even to the fine capillaries, and to this pyogenic infection may be added (ulcerative cholangitis). Stenosis of the common duct, usually at its lower end, results in dilatation of all the bile passages and also the gall-bladder, the mucous folds in the common duct are obliterated (valves of Heister), the wall of the duct is thickened and parts of the dilated system may rupture, oftenest the gall-bladder. The liver necessarily suffers if the condition persists, its cells becoming bile-stained, especially in the outer zones of the acini, followed by cirrhotic contraction of the organ, and at times inflammation of the ducts or areas of necrosis. Of all causes for dilatation of the ducts the commonest is lodgment of gall-stones and this is most frequent in the larger ducts ; if they lodge in the ampulla of the common duct they may produce stasis in both the hepatic and the pancreatic excretion.

Inflammation.—*Cholangitis* is most evident in the large ducts, corresponding to its usual origin by extension from the stomach and duodenum. It may be simple catarrhal swelling and hyperemia of the lining mucous membrane, and in severe grades of this disease the ducts may become partially closed by the edema of their walls, resulting in stasis of the bile-current above, dilatation of the bile-ducts and obstructive jaundice. When the edema subsides the gall flows again into the gut, and the condition is relieved. If chronic, the inflammation may produce dilatation of the main ducts and polypoid growths. Other causes of the disease are irritation by gall-stones, entrance of parasites from the intestine, as round worms, and extension from adjacent structures.

Purulent inflammation of the ducts follows injury by gall-stones, abscess formation near by, especially if breaking into the bile-passages, stasis and disorganization of the bile with pyogenic infection from the gut. It complicates certain general diseases, as typhoid, dysentery and pyemia. The mucous lining of the part is then swollen, discolored, and even necrotic, and the ducts contain yellow or bile-stained muco-pus. The inflammation may pass to other structures, favored by anatomical relations, as to the portal vein, causing pylephlebitis, or to the liver tissue, with abscess formation.

The gall-bladder itself may take part in such a suppurative inflammation or remain for a time uninvolved; it may also be the only part inflamed. The cause of such a local inflammation is most often irritation by gall-stones; dilatation of the part is common, by mucous or muco-purulent fluid; ulcers may form in the wall and open into the peritoneum, the stomach or intestine, or through the abdominal wall. The course of the disease is often chronic and interrupted by periods of apparent recovery. When the gall-bladder is distended with pus it is termed *empyema* of the part.

Cholelithiasis.—Gall-stones are found in about 7 per cent. of all autopsies, but are far commoner in females than in males.

It is probable that they form, like vesical calculi, about a nucleus, which may be only a bit of thickened mucus, less often a foreign body from outside. On this a layer of biliary salts and albumin is deposited, a process favored by stasis of the bile and partial decomposition of its elements, and successive layers are added till a relatively large size may be reached; or many small stones may form. Mutual attrition usually wears facets on the calculi when few and packed together, giving roughly pyramidal and crystalline shapes to them; in size they vary from grains of sand by the hundred to a single stone as large as the gall-bladder will contain. While usually formed in the gall-bladder they may form elsewhere, as in the larger ducts. The color varies from light yellow through different browns to black and their chemical composition may be pure cholesterin, or bile pigment, or the two combined or calcium salts, but most commonly they contain all these elements, and the larger forms are usually in concentric layers.

The results of such formation of biliary calculi may be postponed for years if they lie quietly in the gall-bladder, though there is always a possibility of their causing suppuration, but when they pass into the cystic and common bile-ducts they cause biliary colic, probably the severest pain known, in which death with symptoms of collapse may occur. If not too large to pass into the gut, aided by pressure of accumulated bile above them and gradual slight dilatation of the canal, the escape is proclaimed by sudden cessation of the pain, and such attacks may recur at various periods in the same patient. If they lodge in the course of the outward journey they may cause suppurative inflammation, adhesions, and perforation into the peritoneum or some part of the alimentary canal.

Tumors.—As a general statement all benign and sarcomatous tumors are uncommon in the gall-bladder and ducts. Primary carcinoma seems at times to be associated with calculi of the part though these would probably form in any case from the stagnant bile. In the biliary passages in the liver primary carcinoma may start in the mucous glands of the wall or, after prolonged misdirected hyperplasia, in the epithelia lining the ducts. A common site of the neoplasm is about the lower end of the common duct.

Icterus.—A symptom common to many diseases of the liver and its ducts is staining of the organ and of various tissues of the body with biliary pigments. This is known as *jaundice* or *icterus* and is usually described as of two varieties, hematogenous and hepatogenous, as if bile pigment were formed in the circulating blood as well as in the liver. The opinion now is that the only source of bile pigment is the hepatic cells and hence strictly there can be no hematogenous jaundice; and yet there are cases where general yellowness of the tissues occurs with no trace of mechanical obstacle to the flow of bile, as is proved by the presence of bile in the feces during life and no apparent hepatic lesion after death. Such icterus is common in certain infectious diseases, certain intoxications, and occurs in the new-born as icterus neonatorum. Such icterus may be produced experimentally by injection of various substances into the blood-vessels of animals, as frozen blood, toluylendiamin, etc.

These cases of icterus may be explained as the result of disordered innervation, of access of toxic substances to the liver, or of disordered flow of bile formed in the hepatic cell, whereby it reaches the capillaries of the lymph system instead of the biliary. The name *paracholia* may be given to these unexplained cases of icterus, and among them may be grouped those which follow chloroform, phosphorus and serpent poison, pyemia and other infectious diseases, and some cases of icterus neonatorum; though in the latter case mechanical alterations of the circulation in the new-born and edema of the periportal tissues have been supposed to be causal.

Hepatogenous jaundice in the old sense, also called obstructive and resorptive, occurs when there is an obstacle to the flow of bile into the intestine, and the obstacle may be in the papilla, the main ducts, as in catarrhal jaundice, associated with catarrhal inflammation of the duodenum and main ducts, or it may be within the liver from stenosis of the capillaries, inflammation of their walls, and various compressions. Obstruction of the large ducts by inspissated mucus, gall-stones, round worms, echinococcus cysts and pressure from neighboring organs will explain other cases. In some instances thrombosis of the portal vein, loss of motion in the diaphragm and severe emotional crises may cause it.

Since experiments indicate that icterus occurs only so long as the lymphatics of the liver remain patent, all varieties appear to result from a diverted bile circulation; to which dilatation of the bile capillaries directly tends since it loosens the hepatic cells and facilitates entrance of the bile into the perivascular lymph spaces. The liver normally has an internal secretion, by which urea and other matters are passed into the blood, and bile also may enter the circulation when its excretion is impeded.

The skin, the sclerotics, the lining of the arterial system, the mucous membranes generally, all normal secretions and pathological exudates, and all parts of the body except the teeth, the hair and the glans penis may be tinged with the icteroid hue, varying from light yellow to darker shades with greenish admixture. In certain cases the feces are nearly colorless, clay or putty colored, but as their color is due to the presence of both the biliary and the pancreatic secretions bile may not be absent even from light-colored feces if the pancreatic juice is deficient. An important train of nervous symptoms results from jaundice, probably due to the presence of biliary acids and salts in the circulation rather than to the pigments.

Peritoneum.

Malformations.—Imperfect development of parts of the peritoneum have been described, especially of the omentum; excessive length of the mesentery permits volvulus, and other folds, if too long, allow the organs to which they act as ligaments to fall to lower levels (enteroptosis). Fissures in the serous folds may allow portions of the gut to pass through (hernia), and similar results occur in pouches on the abdominal walls.

Disorders of Circulation.—*Active hyperemia* is observed in the first stage of peritonitis, general or in scattered areas, and about tubercles and tumors there may be a zone of congestion. *Passive hyperemia* follows from obstacles to the portal circulation and in long-standing cases the vessels may become much dilated, especially the veins along the line of reflection from abdominal organs to walls, as in cirrhosis and tumors of the liver.

Slight *hemorrhage* into the peritoneum or beneath it may occur in portal obstruction, phosphorus-poisoning, death by asphyxia and some other conditions. Large collections of blood in the cavity result from rupture of organs (spleen and liver), of aneurysms, injuries and operations on various organs, tubal pregnancy, typhoid and other intestinal perforations. The blood collects in dependent parts of the cavity and may be fluid or coagulated; even large quantities may be absorbed and the corresponding lymph nodes are deeply colored with it.

Serous accumulations in the abdominal cavity (ascites, dropsy) occur without local inflammation in general hydremias, as in chronic nephritis, valvular heart disease and cachexia ; in other cases the peritoneum alone contains fluid, as when there is marked and prolonged portal stasis, commonly observed in atrophic cirrhosis. With inflammation and local diseases such collections are found in tuberculosis of the peritoneum and tumors, with diseases of the thoracic duct, the fluid is chylous. The ascitic fluid is usually clear, bile-stained in some cases, mixed with flocculi of fibrin, varying in amount from a few cc. to many liters, distending the cavity, pushing the diaphragm and organs upward and causing dyspnea, producing white scars in the corium from rupture of its fibers. When removed by tapping the ascitic fluid may renew itself many times. Adhesions may localize it and cause sacculated dropsy, and if between the layers of the omentum it is called omental dropsy.

Injuries of the peritoneum are dangerous from exposure of the membrane to the air, contact with rough foreign substances, and access of bacteria; under aseptic conditions and supplied warmth and moisture surgical wounds are almost without danger to the healthy peritoneum. Injuries which involve the alimentary canal also, with exit of its contents, are especially liable to end in severe inflammation.

Inflammation.—The peritoneum has a superficial extent about equal to that of the entire surface of the body, it is in constant movement, both in the layer lining the anterior wall and in the visceral layer, its power to absorb various fluids is enormous, its vascular and nerve supplies are abundant and it is in close relation to important organs, vessels and nerve centers: hence a general inflammation of the membrane is necessarily of great clinical importance.

While primary idiopathic peritonitis may occur, in the vast majority of cases the inflammation follows access of pyogenic bacteria, either through the blood-vessels, which is infrequent, or by extension from organs already inflamed, or by perforation of certain parts, especially of the intestine. The first may occur in pyemia. The second arises in many ways but the only normal opening from the peritoneum to the outside world, the Fallopian tube, is worth specially mentioning in this relation because of the frequency of peritonitis in the female, from gonorrheal and other salpingitis, puerperal infections and other genital conditions. The third variety follows strangulated hernia, ulcer and perforation of the gut, abscess and tumors and injuries of any of the abdominal organs.

Peritonitis is described as localized when but a small part of the membrane is affected, or general when most of it is attacked; either may precede the other, for a local inflammation may become general and the latter may subside and leave a chronic circumscribed process. The area attacked becomes actively hyperemic, loses its luster, and soon is the seat of an inflammatory exudate. This may be serous or purulent from the first, but is often fibrinous, and the effect is to bind together adjacent surfaces and so limit the extent of the disease; later the fibrin is liable to become purulent and if capsulated an abscess is thus formed in a pocket between certain coils of intestines; if this remains, the pus may thicken and become absorbed, fibrous tissue and lime salts completing the healing process. At times the pus burrows for long distances, and is voided through some hollow organ or externally. If the abscess rupture into the rest of the cavity a general and rapidly fatal peritonitis may result, as is also the usual case when the amount of irritating material is large or very septic, as with strangulated hernia, multiple bullet wounds of the gut, or rupture of the gall-bladder. In such cases the lesion may deserve the name of gangrenous peritonitis. A hemorrhagic variety is also described, which is merely an expression of such local or general conditions that extravasation and hemorrhage easily occur, as in scorbutus.

Chronic peritonitis may follow the acute form, especially when encapsuled, or it may be limited in extent and of slow type from the first. A form of this variety is at times seen in the upper zone of the abdomen, resulting in thickening of the capsule of liver and spleen. The thickened membrane has been thought to resemble sugar after melting and hardening, and has received the German name suggesting this of "Zuckergussleber," another instance of the unfortunate choice of pathological terms from the diet list. Chronic peritonitis is common in the pelvis, especially of women, and results in organized bands of adhesion between the contents of the cavity and of the abdomen proper.

Infectious Granulomata.—The only important disease of this kind affecting the peritoneum is *tuberculosis*, which is very rare as a primary infection but common as a sequel of similar disease in the alimentary tract, its lymph nodes and other organs. It may occur as a general miliary tuberculosis of the entire membrane; it may be limited to small miliary masses on the serous coat corresponding to ulcers on the mucous coat of the intestine; various groups may coalesce and form large masses of thickened tuberculous tissue, or it may produce increased thickness of the omentum and various adhesions between intestinal coils so that it is impossible to unravel the abdominal contents. Copious transudate of serous fluid accompanies some forms of peritoneal tuberculosis, which is always a chronic process and the fluid, either sacculated or free, in some cases may be reabsorbed and the process end in apparent recovery.

Tumors.—Chronic peritonitis is at times accompanied by the formation of many small fibromata (or endotheliomata) over the serous surface, giving the effect on first sight of tuberculosis. Lipomata develop in the omentum quite frequently, fibroma and fibrosarcoma of the broad ligaments are also found at times.

Primary carcinoma is rare, though it may take origin in fetal remnants, as of portions of the alimentary tissues constricted off during development. Colloid or gelatinous carcinoma occurs with similar disease of the caput coli, rectum, etc., and at times is diffusely spread over large areas as miliary or larger nodules. Similar conditions occur in general carcinomatosis. Other organs from which carcinoma may invade the peritoneum are the uterus and ovaries, stomach and liver, and even the pleural contents by extension through the diaphragm. In all forms a certain amount of serous exudate may be found, at times hemorrhagic, and various adhesions between the viscera are common.

Parasites.—There seem to be no strictly peritoneal parasites, but distomum hepaticum and echinococcus from the liver, filaria from the blood and lymph channels, actinomyces from the cecum and the kidney, and a few others have been found.

CHAPTER VI.

THE URINARY SYSTEM.

Kidney.

Malformation.—In viable infants one kidney may be lacking, usually the left, and the right, often provided with two ureters, is then large enough to perform the entire renal function. The division into lobes, normal in the fetus, often persists to adult life. Extra kidneys are uncommon. The two organs may be united at the lower ends by fibrous or renal tissue, forming a horseshoe kidney, the pelvis usually lying on the anterior aspect of the double organ with anomalies in ureters and vessels. United kidneys, and either one singly, may lie below the normal level, at the pelvic brim or even within the cavity.

As an acquired dislocation the right kidney falls forward and downward from looseness of its ligaments, less often the left, which is more liable to congenital displacement; the cause of such a floating kidney may be pressure of the mass of the liver, owing to right-sided pleural distention, or pressure from without, and at times traction from the stomach and transverse colon appears to have the same effect. The motion takes place along an arc of which the renal vessels form the radius, and is of considerable extent when the perirenal fat is deficient. As the condition tends to become more marked the radius lengthens, the range of motion increases, the nerves and vessels, the ureters and other structures, are dragged upon and severe symptoms develop. Among these are pyelitis and hydronephrosis, intense pain, and pressure effects.

Disorders of Circulation.—*Anemia* of the kidneys occurs with general anemia from any cause, and at times, even when the organ was probably hyperemic during life, it appears bloodless on autopsy; this may be due to the swelling of the epithelium in the cortex, edematous or from post-mortem coagulation, which mechanically drives the blood out. The glomeruli may appear as dark-red spots on a pale background, for their arterial tufts remain full. With true anemia the whole organ is pale and small.

Hyperemia.—The kidney is the one organ in which the difference between active and passive hyperemia is apparent after death, owing to the peculiarity of its circulation. Arterial hyperemia seldom occurs without added signs of degeneration or inflammation. It may be found as the result of irritating substances excreted through the kidney, as cantharides, carbolic and oxalic acids, and in scarlet fever and extensive superficial burns, when death occurs before inflammation develops. The kidneys are then equally affected, large, succulent, the capsule strips off readily (in the absence of previous disease), the cortex is swollen and edematous, dark red, and markings often very distinct, glomeruli distinguishable on gross inspection, the pyramids less congested.

Passive congestion commonly results from general circulatory disorders, valvular heart disease, emphysema, fatty degeneration of the myocardium and similar conditions, though obstruction of one renal vein will produce stasis in the corresponding kidney, and of the vena cava, in both. In an acute form renal stasis occurs with death from asphyxia. When simple, passive congestion results in a large kidney, capsule not adherent, juicy, surface smooth, venous stellulæ prominent, cortex swollen and dark red, glomeruli prominent (a hand magnifying glass may be needed), pyramids also dark or brownish-red. This condition of stasis, like that of active hyperemia, seldom lasts any time without resulting in degeneration of the epithelia or increase of fibrous tissue or both.

Hemorrhage in slight degree may follow severe congestion of either form, enough to appear in the urine as individual red cells, and may usually be considered as simple diapedesis. Injury of the organ, purpura and hemophilia, and tumors may be accompanied by larger bleedings when affecting the kidney. With infarcts hemorrhage is common.

Thrombosis of small renal veins may occasion but little disturbance; when the larger branches are affected the kidney becomes congested and edematous and fatty degeneration occurs in the part involved.

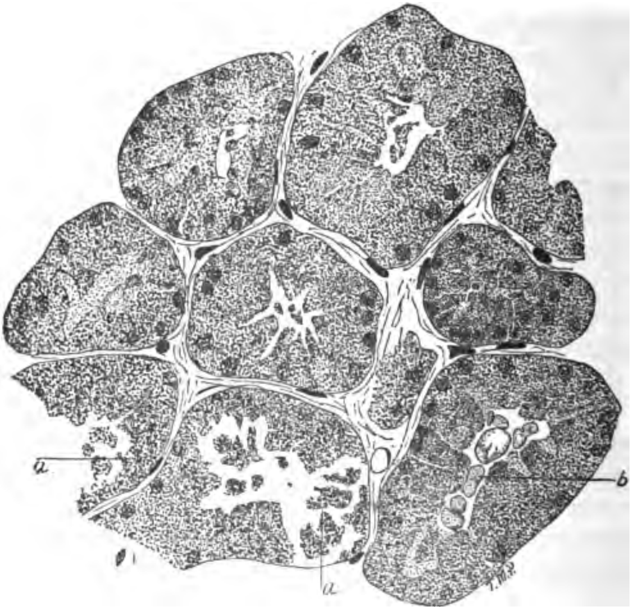
Embolism is more common and more important, for the renal arteries do not anastomose. The result of embolism is anemia of the part supplied in the usual wedge- or cone-shaped form, with the base superficial. Red infarction is less common in the kidney than the anemic form. The usual steps toward healing, death and resorption of the cells, invasion peripherally by new vessels and connective tissue, scar formation and contraction, follow rapidly and the capsule may be adherent over an area of firm, yellowish, depressed fibrous tissue, the site of the infarct. Such conditions may be multiple and not limited to the surface of the kidney. Other forms, leading to suppuration, will be treated under a later heading.

Atrophy.—Loss of function, diminished blood supply and pressure upon the kidney lead to atrophy of its functionating epithelia; hence it accompanies old age as a senile change, and is the usual result of damming back the flow of urine so that the organ is distended from within. In old age the atrophy may affect the cortex almost entirely, especially the subcapsular layer; the surface is then granular, or rather it presents a number of fine depressions, and the epithelium corresponding is degenerated, pigmented or lost, the glomeruli are small and their capsules thickened. If the whole organ is involved it is small, tough and pale or brownish. A replacement fibrosis may be added and the organ is distorted by the fibrous contraction. This form is closely associated with arteriosclerosis, usually general throughout the body, at times manifested chiefly in the kidney. All parts of the organ may not be involved, but here and there the thickened vessels produce areas of atrophy and shrinking, often with lime salts deposited in them, and the urinary tubules, being compressed while excretion goes on above, dilate and form little retention cysts, both on the surface and through the tissue. The capsule may be adherent, the surface is always granular over these areas. Such arteriosclerotic atrophy of the kidney is common in gouty patients, after chronic lead-poisoning, and in the syphilitic. Similar effects follow endarteritis obliterans of the renal vessels, as so often associated with chronic inflammation. Hence a series of kidneys might easily be collected illustrating these various conditions and combinations of them and transitions between them, so that for purposes of description artificial boundaries have to be assumed which in practical experience are seldom noted.

Hypertrophy of one organ follows degeneration and disease of the other as a functional and compensatory change. It may be congenital, when one kidney is cystic or the seat of a tumor, it may be acquired late in life when a kidney is diseased, and is of great practical importance in surgical removal of the organ. The one hypertrophied presents both an increase in the size of its elements and also in their number and hence is a combined hypertrophy and hyperplasia. The general condition of the patient and the constitution of the urine may be perfectly normal with such compensatory hypertrophy.

Degeneration.—*Acute renal degeneration* is usually of the albuminous variety and as "cloudy swelling" accompanies many febrile diseases and the action of certain poisons and irritants. Among the former, typhoid and scarlet fevers, diphtheria and septicemia, pneumonia and others commonly result in cloudy swelling of various organs, especially the kidneys, and the explanation is probably that their toxins are excreted (as well as the bacteria) through the organs and poison the cells by chemical action on their protoplasm. This resembles the action of arsenic, mercury and phosphorus, cantharides and other substances more commonly known as poisons. In slight degrees the effect of such action is to cause the epithelial cells to swell and become opaque, from the presence of fine or coarser granules in them; congestion and slight edema commonly accompany this condition. More pronounced action leads to fatty degeneration and coagulation necrosis of the epithelia.

FIG. 22.



ACUTE PARENCHYMATOUS DEGENERATION OF THE KIDNEY. (*Delafield and Prudden.*)

a, epithelial cells, degenerated and desquamated, the nuclei preserved in places; *b*, hyaline material in the lumen of the tubes.

Acute nephritis may follow or accompany the effect of such toxic influence, as is well illustrated by the usual phenomena in scarlet fever, where with a mild case and in the beginning degeneration occurs, later acute exudative nephritis, and still later, often after the fever has disappeared, acute productive nephritis; apparently these three degrees correspond to the intensity of the toxic action, the cells reacting but not dying with slight degrees, degenerating with stronger doses, and the effect passing over to the stroma with the highest degree of influence. The gross appearance of the kidney in simple cloudy swelling is slight increase in size and succulence, the capsule strips readily, the cortex is swollen and pale and its cut surface is opaque as if dashed with hot water. It may also be moderately congested.

Fatty Degeneration.—Fatty infiltration occurs only where fat is normally present; as this is not the case in the human kidney a fatty degeneration is the only change of this character which can be described. It follows the albuminous form both in febrile cases and in poisoning, especially with phosphorus, and may occur with no sign of inflammation, either exudative or productive, as in anemias of various kinds. It occurs in cachexia, as with chronic tuberculosis, and is a regular attendant upon inflammation both acute and chronic; the element common to all these is defective nutrition of the epithelia with or without added toxic action.

The fat may be limited chiefly to the region of the convoluted tubes, and if absorbed the result may be minute depressions over the cortex. When general, fatty degeneration produces a large pale kidney with a decided yellow look unless there is much congestion with it. The fat is usually visible to the naked eye as minute yellow points in the course of the urinary tubules, either in the cortex, where alternating blood-vessels and fatty tubes give a peculiar distinctness to the markings, or in the pyramids, where the long collecting tubes radiate like fine yellow lines on a darker background, or in both places. Such general fatty degeneration is not uncommon in the later months of pregnancy. When nephritis coexists the gross and microscopic appearances correspond, as will be mentioned in discussing nephritis.

Amyloid Degeneration.—The causes of this variety are those of all amyloid change, suppuration in bones and in the lungs, as common in tuberculosis of these organs, syphilis, and other cachectic conditions, but the kidney seems especially liable, being affected when other organs escape. The organ may be large and yellowish, firm to the touch, friable, pale or mottled, and usually the gross appearance is a combination of fatty and amyloid degeneration. The capsule may be adherent, the surface usually is smooth, the cortex increased in size and marked by grayish lines, corresponding to the altered vessels, and yellow spots of fat. The application of iodine makes the amyloid portions prominent.

In other cases the change is limited to the vessels or the glomeruli and can be detected only on microscopic examination. The slighter degrees are not accompanied by widespread fatty change because the vessels may still be open enough for the nutrition of the epithelia; these cells are not directly involved in the amyloid change but the membrana propria may show evidences of it. Clinically the results of amyloid degeneration are oliguria, albuminuria, general edema and nervous symptoms with coma or convulsions.

Glycogen and fat together occur in the kidneys of diabetics, especially in the cells of Henle's loops, and in kidneys hardened in absolute alcohol the addition of iodine develops the characteristic brownish-red color.

Lime salts occur in the form of "infarction" of the urinary tubules, and in the new-born are taken to signify that the child has breathed (their absence does not prove the opposite). Similar deposits occur in glomeruli which have atrophied, in degenerated epithelia, and accompany various inflammatory and toxic conditions.

Inflammation.—The importance of the renal function, the certainty that toxic substances in the blood will sooner or later reach the kidney, the rapidity with which irritation leads to degeneration and inflammation, the liability of the organ to both active and passive hyperemia, and the great resemblance clinically between various diseases of the part, have together produced a confusion in the pathology of the kidney which makes the subject particularly difficult to present or understand. The name nephritis, with qualifying adjectives, has been given to congestions, degenerations and inflammations, so that it resembles the expression "Bright's disease" in conveying more than one idea. It is still possible to arrange the lesions of the kidney in an orderly manner and to remember that they tend to combine and to pass from one into the other, and that all non-inflammatory lesions may become inflammatory, and that any acute form of nephritis may become chronic if the patient lives long enough.

Etiology.—Injury, severe chilling of the surface, burns and scalds, ingestion of poisonous and irritating substances, and many bacterial toxins may cause inflammation of the kidney, which, as distinguished from the more common clinical types, might be called primary.

As a general rule, however, nephritis is associated with some other lesion. This may act by circulatory disturbances, as in lung and heart disease; or by the excretion of toxic substances formed in the body, as seen in connection with long-standing intestinal dyspepsia and absorption from the decomposing contents of the gut; or perhaps general conditions of malnutrition, as in tuberculous, syphilitic, cancerous, anemic and other cachexias; in many other cases we may assume the action of a toxin derived from one or more bacteria, with presence of the microörganisms in the kidney itself (embolism by pyogenic cocci, anthrax, etc.), or excretion of toxins alone. Among the poisons arsenic, phosphorus, carbolic and oxalic acids, mercury, lead, cantharides and turpentine may be mentioned. Among the diseases with the presence of microörganisms in the kidney may be mentioned pyemia, mycotic endocarditis, malaria, anthrax, pneumonia, typhoid fever, and various suppurative diseases due to pyogenic organisms, cocci, b. coli communis, b. pyocyaneus, gonococci, and others. As an example of toxin alone (probable in many cases), cholera, diphtheria, variola and inflammatory rheumatism may be mentioned.

The locality most often involved is that of the convoluted tubules and the glomeruli; indeed the latter may be so nearly the only part affected as to justify the term "glomerular" nephritis, as in cantharides-poisoning. In other cases, especially the chronic forms, no element of the organ escapes. Again, as a rough rule, it may be stated that in the acute forms of nephritis without suppuration it is the epithelium which especially suffers, while in subacute and chronic inflammations the fibrous stroma presents the chief lesions and the epithelia suffer in consequence of these.

The various forms of nephritis are:

- Nephritis
  - acute
    - exudative
      - catarrhal.
      - hemorrhagic.
      - suppurative.
    - productive—diffuse.
  - chronic
    - exudative — large white kidney, chronic diffuse nephritis.
    - productive—cirrhotic kidney, chronic interstitial nephritis.

Acute Exudative Nephritis.

Synonyms.—Acute Bright's, parenchymatous, glomerular, catarrhal, or tubal nephritis.

In acute exudative nephritis the exudation may consist, as elsewhere, of serum, fibrin and pus, with diapedesis of red cells, and the resulting type of the disease depends in great part on which of these is most abundant. If serum predominates, though both red and white cells may be present and fibrin also, the form often called catarrhal or desquamative results. Congestion regularly precedes and accompanies all varieties. The glomeruli may be chiefly affected or the tubes or both, and during life the urine contains serum, casts and relatively few leucocytes and red cells. This form may follow exposure to cold, and often accompanies pregnancy and infectious disease, especially scarlet fever. The epithelia may be the seat of cloudy swelling or of more serious degeneration, but the essential pathological element is the condition of the blood-vessels which permits exudation, of serum especially.

The organ is swollen, capsule not adherent, surface smooth and congested or pale or mottled, succulent, cortex swollen and cloudy, glomeruli at times very prominent. Microscopically the discoverable lesions may be out of all proportion to the clinical gravity of the case; there may be only a few casts in the tubules with evidence of congestion, and in the glomeruli distended arterial tufts and desquamation of the lining cells. Severe cases show more marked lesions, the epithelia in the cortex may be swollen, compressed, detached from the wall, or degenerated; the tubes are distended with (probably) altered fibrin and may contain red cells beside epithelia and leucocytes; in the urine these appear as hyaline casts and separate cells, with albumin. In the glomeruli the capillaries may be congested or not, but the covering and lining epithelia are swollen, desquamated and degenerated more than in the milder cases, even to complete filling of the capsule of Bowman. In the cortex here and there small collections of white cells may be found which differ from abscess formation in the preservation of the cells and the absence of bacteria.

Acute Hemorrhagic Nephritis.—While the former variety may be fatal in a comparatively few hours, it is possible to recover from it without serious damage to the kidney tissue, and this is the rule in mild cases. When the intensity of the producing cause is such as to result in great numbers of red cells escaping from the vessels, as in malignant endocarditis and scarlatina, some cases of carbolic and other irritating poisons, the gravest form of infectious or toxic acute nephritis occurs. The kidney is usually very large, soft, congested or pale, but marked everywhere in the cortex by smaller or larger points of extravasation, of a deep red color, corresponding to the hemorrhages. With infection these areas may be entering upon suppuration.

While this is but one variety of exudative nephritis it differs from the foregoing in being uniformly fatal.

Acute Suppurative Nephritis.—This condition, which is marked by exudation of pus-cells and the formation of abscesses, may be due to extension of inflammation from the pelvis of the kidney (ascending suppurative nephritis) and this form will be treated under pyelitis.

It may also be connected with pyogenic organisms in the circulation, as in mycotic endocarditis, infectious osteomyelitis, pyemia and other distant diseases with suppuration. In a chronic form it is often associated with tuberculosis of the kidney. The microörganisms are the pyogenic cocci, b. coli communis, and as perhaps a casual, perhaps an accidental find, typhoid and pneumonia organisms. The first step in the process is embolism of the renal capillaries by the bacteria with necrosis of the cells and a zone of reactive inflammation. These areas rapidly become small abscesses and the organ may be dotted with them, or they may coalesce and form large cavities. The pus finds its way out by the ureter or burrows into the perirenal tissue or remains in situ. Usually both kidneys are affected but even then one may show far more damage than the other.

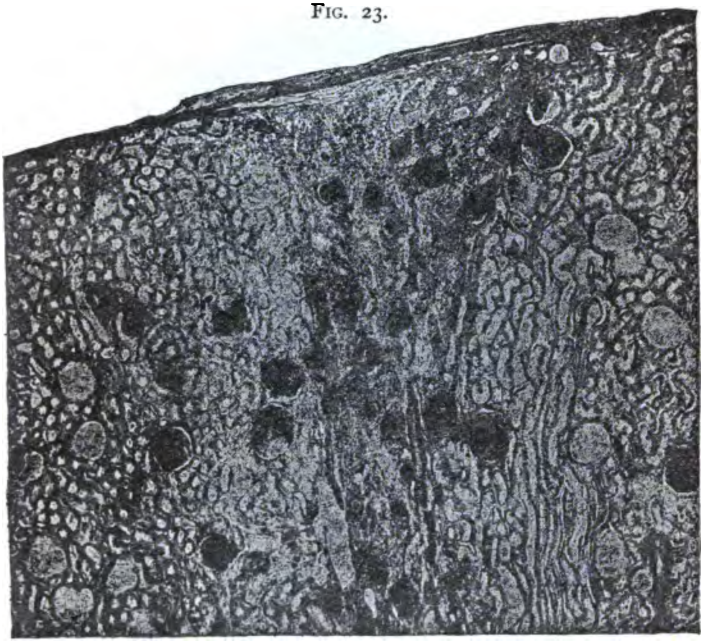
As a result of injury, part or the whole of a kidney may be crushed, with the production of one or more small purulent foci or conversion of the whole organ into a mass of pus and broken-down tissue.

Acute Productive Nephritis.

Synonyms.—Subacute diffuse nephritis, "wedge" kidney.

This form of acute nephritis might be called exudative-productive, for its histology is a combination of both characters. Even when death occurs early in the disease the productive change has already begun. In many cases the distribution of the new fibrous tissue is along the arteries running to the cortex. This results in the formation of long slender cones about the vessels, their bases on the surface, which is depressed by their contraction. These are usually termed "wedges," but this view takes in only two dimensions while the development of new tissue occurs in three, the mistake arising from the fact that the organ must be cut open before the change is noted and then a flat surface presents. Such fibrous increase may occur also scattered through the tissue. In the older cases this new tissue is very poor in cells. The other microscopic changes are swelling or degeneration and atrophy of the epithelia, formation of casts, desquamation of the glomerular lining with at times marked hyperplasia of these cells and pressure upon the arterial tufts.

FIG. 23.



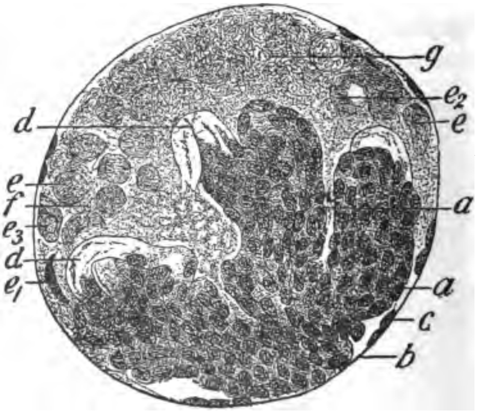
ACUTE PRODUCTIVE NEPHRITIS. (*Delafield & Prudden.*)

In the middle a conical "wedge" of fibrous tissue about an interlobular vessel. Epithelia in this area lost. Glomeruli here are atrophic, elsewhere many have fallen out of the section.

Moderate exudation is present early; later there may be but slight trace of it. Lymphocytic infiltration may be profuse.

The gross appearances of such kidneys are increase in size, non-adherent capsules, smooth red or mottled surface, cortex marked by the "wedges" described above with dimpling of the surface over them; these occur also in chronic nephritis.

FIG. 24.



PROLIFERATION AND DEGENERATION OF GLOMERULAR EPITHELIUM. (*Ziegler.*)

a, normal capillary loop; *b*, capsule of Bowman; *c*, epithelia of capsule; *d*, vascular loops denuded of epithelia; e_1-e_3, desquamated degenerated glomerular epithelia; *f*, *g*, exudate between the cells.

The results of acute productive nephritis are always worse than those of the exudative form if the patient survives the first attack, for the kidney is permanently injured by a fibrosis which like the same process in other organs has a tendency toward persistent increase. Attacks of this kind are common after diphtheria, scarlet fever and with pregnancy.

Chronic Nephritis.

The essential element in either form of this lesion is the marked fibrous increase in the stroma of the organ with consequent changes in the epithelia, but in some cases there is an inflammatory exudate (serous) which appears in the urine in relatively large quantity and persistently, and with this the case is remarkable clinically for general dropsy. While the lesion then is productive, the exudation distinguishes the disease from the pure chronic productive nephritis in which there is no albuminuria, or only an occasional appearance of this symptom, and anasarca is commonly absent.

In **chronic exudative nephritis,** as in the other form, all the elements of the organ are involved. The cause may be a preceding acute nephritis, or chronic passive congestion of the organ, and this factor enters in with any cases which complicate pulmonary tuberculosis, emphysema, valvular cardiac disease. Other cases follow syphilis, lead-poisoning, gout, alcoholism, and in the latter there is often hepatic cirrhosis as well. In the young, nephritis of this type may develop after scarlet fever or without apparent cause.

The gross appearance of the exudative form of chronic nephritis varies, but commonly the organs are enlarged. In color they may be white from combined anemia and fatty degeneration but they may be mottled or dark red in proportion to the congestion present. The capsule in many instances is not adherent and the surface is smooth, in kidneys of smaller size the capsule may be so adherent that little grains of kidney tissue are torn off with it and appear on its inner aspect in scattered groups. Larger masses of tissue may be torn off where a vein passes between capsule and organ, but this will hardly be confused with the dotted groups mentioned.

In the smaller forms of kidney, when the fibrous tissue has contracted a great deal, the surface of the organ is often marked by small cysts with clear pale contents or thicker and dark yellow or green fluid, or the former site of such cysts may be marked by little puckered scars. These retention cysts are more common in the next variety. The cortex may present but little change beyond swelling and paleness, or it may be congested and at times dotted with punctate hemorrhages. In other cases the fibrous cones are well developed and scar-like bands of tissue run from depressions on the surface into the organ. The markings of the cortex are commonly not well preserved.

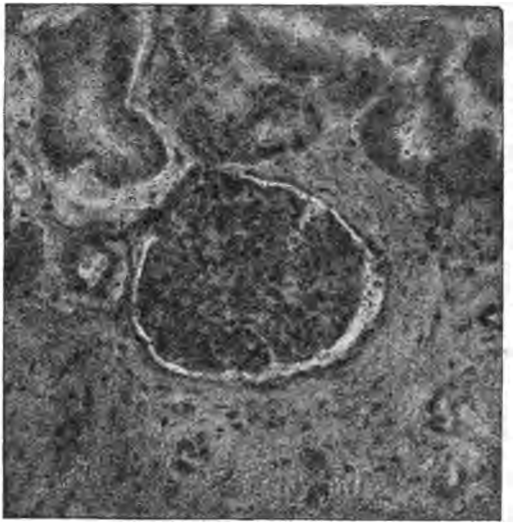
Microscopically the appearances differ with the age of the process. In recent cases, where death has occurred fairly early, there is well-marked and general degeneration of the epithelia with scattered foci of new fibrous tissue, rich in cells, which may communicate by fibrous processes. Spindle cells and delicate fibrils may be found. The process is specially developed about the blood-vessels and the glomeruli. In the latter the capsule is thickened, the lining cells are increased in number and degenerated, but many glomeruli appear nearly normal. While the main effects of the process are most apparent in the cortex the increase in the stroma and the epithelial degeneration may be found in the pyramids also. The urinary tubules may be atrophic, filled with degenerating epithelia and coagulated fibrin, or dilated; both red and white blood-cells may occur in their contents. In the glomeruli some of the capillaries may be the seat of amyloid changes as also other vessels; a certain degree of arteritis is common, with thickening of one or more coats and diminution of the lumen.

Chronic Productive Nephritis.

Synonyms.—Chronic Bright's, interstitial nephritis, cirrhosis of the kidney.

In this form the rule is that the kidneys are very atrophic; they may weigh together much less than the single normal organ; rarely they are larger than normal. The capsule is adherent, often stripped off with difficulty, and the fatty capsule may be increased to make up for the loss of volume in the atrophic organ. The surface is very granular, frequently presents cysts which are found also in the interior, the cortex is irregularly thinned and its markings lost on gross examination. The new fibrous tissue is irregularly disposed through both cortex and pyramids; the pelvis may be widely dilated when the nephritis follows obstructed flow of urine, or in a condition of chronic catarrhal inflammation.

FIG. 25.



CHRONIC INTERSTITIAL NEPHRITIS. (*Delafield & Prudden.*)

Marked fibrous hyperplasia about the glomerulus, which is also thickened. Epithelium degenerated, cell bodies granular, nuclei absent in places.

Microscopically the glomeruli, vessels and tubes present the same atrophic and degenerative conditions as in the former variety. In other words, so far as the histology of chronic nephritis is concerned all forms might be described together; the distinction made here is justified by the differences in the clinical course of the two, and each may be recognized by the symptoms.

With the latter form hypertrophy of the left ventricle is usually more marked than with the former, not due so much to the changes in the kidney as to the general arteriofibrosis throughout the entire system of arterioles, which multiplies the work of the left side of the heart both by increasing the friction against which it has to labor and also by depriving it of the vascular recoil or peristaltic wave which takes up the charge received after each systole and carries it to the capillaries without additional tax upon the heart. The retention of water in the body because of lessened urinary excretion, and the retention of various chemicals which act as irritants to the heart have also been held to explain the cardiac hypertrophy and the pulse of high tension in these cases. The result in the heart is so large an increase in its tissue as to warrant the name *cor bovinum*. It is in this form that hemorrhage into the retina or the brain is especially common, as might be expected from the combination of degenerated arteries and powerful systole.

Uremia and its explanation have been mentioned in Part I., p. 57.

Cysts of the Kidney.—Dermoid cysts rarely occur. Small retention cysts are common in senile atrophy and in chronic interstitial inflammation. Occasionally a kidney, normal in other respects, presents a single fairly large cyst in the cortex; probably due to closure of tubules by mineral matter (calcium salts) with local inflammation and stenosis of the tube. In some cases an entire kidney is converted into a multilocular cyst as the result of calculi in the opening of the ureter and consequent dilatation by accumulating urine behind the obstacle; the kidney tissue may be recognized here and there as a thin peripheral film.

A similar condition occurs, usually in one kidney, associated with atresia of the papillæ, as a congenital anomaly.

Tumors.—Fibroma, lipoma and leio-myoma sometimes occur in the kidney; the first may attain a large size, the others are usually small. Sarcoma of the organ is rare but myo-sarcoma, with striate cells, has been described.

Hypernephroma is usually benign if small, and malignant when larger; it starts in adrenal tissue included in the kidney during fetal life. The tumor corresponds in its structure to that of the adrenal body but the relative proportion of cortex is apt to be larger and its blood supply is usually rich (capillaries); bleeding and cavernous forms are not uncommon, recognizing that hypernephroma is in itself a rare tumor, and as in other neoplasms of the organ, glycogen is frequently present. The malignant forms cause metastasis to various organs owing to the ease of transmission though the dilated capillaries, and with malignancy the normal adrenal structure gives place to irregular atypical cells and newly formed vessels with degenerated (hyaline) walls. *Adenoma* may start in hyperplastic areas of renal cells, as in similar hepatic conditions, and this form of tumor may become cystic. *Adenosarcoma* occurs in early life and often becomes malignant after a period of latency. Primary *carcinoma* is not common in the kidney; it occurs in late adult life and in most cases begins in the epithelium of the cortical tubes. Metastases are not so common as in carcinoma of other organs. Secondary tumors of this variety frequently occur in the kidney, usually betraying their embolic origin as minute scattered nodules in the cortex.

Infectious Granulomata.

Primary *tuberculosis* of the kidney is very rare. As a secondary lesion it occurs in general miliary disease and with tuberculosis of the urinary and genital organs of the pelvis. The spread of the disease is usually upward, against gravity, though when the patient is lying down gravity is negligible, rarely it begins in the kidney or its pelvis and spreads down; and the entire urinary system, kidney, ureter and bladder, is commonly affected together. The disease is more common in males.

The kidney presents one or more, usually several, tubercular abscesses, with caseous contents or empty, and clothed with a pyogenic membrane. Corresponding to the upward course of the disease, the pyramids may be nearly all replaced by such abscess cavities while the cortex shows but little change, or the surface of the organ may contain miliary tubercles and scar tissue. Later on the entire interior of the thickened capsule may be filled with thick pus and caseous material. The lesion may be the only tubercular process, or pulmonary disease may also exist. What kidney tissue remains may be congested or in a condition of chronic inflammation or atrophy. If one kidney is involved the other may be the seat of chronic nephritis by the time the case comes to examination, but as the course of the disease is very slow, probably the other organ may be fairly healthy for long periods.

Syphilis.—Congenital syphilis may be associated with obliterating inflammation of the renal vessels and atrophy of the glomeruli, and similar effects with amyloid changes may accompany the acquired disease. Gumma in the kidney is rare but at times thick, stellate scars in the cortex appear to have had that origin.

Parasites.—Beside the bacteria which may be excreted through the kidney or lodged in it, oïdium albicans and actinomyces among the vegetable parasites and echinococcus, cysticercus and pentastomum among the animal, have been noticed. In tropical countries the filaria sanguinis occurs in all parts of the organ, chiefly in its dilated lymph channels, and causes the condition known as chyluria. In these cases there may be chronic nephritis and a peculiar opaque and greasy look in the pyramids, with dilated and varicose lymph channels.

Calculi, Pigmentation, Etc.—In the new-born, from the second to the fourteenth day, collections of uric acid and urates occur in the large tubes of the pyramids; a similar condition is not uncommon in the adult kidney with gout, and lime salts are found here in diseases of the bones. Biliary and malarial pigment may be found in the renal capillaries; disorganized hemoglobin remains after hemorrhages; ingested stains, like silver, more commonly affect the cells than the stroma of the kidney.

Calculi form in the pelvis of the kidney as fine sand, which passes out almost without symptoms, or larger sizes which cause severe attacks of renal colic, and yet larger stones with many projections which produce distention of the kidney, atrophy of its tissue, and reach up into the various dilatations of the calyces. They are of the same chemical composition as the vesical calculi; see p. 220. Their effects on the kidney are partly due to irritation in the pelvis, which at times appears to be connected with the development of carcinoma, and partly due to the destruction of the kidney from damming back the flow of urine.

Perinephritis.—In the fatty tissue about the kidney contusions and other injuries may produce a suppurative inflammation, but more commonly this attacks the part by extension from caries of the vertebræ or inflamed retro-peritoneal lymph nodes. A distinction is made between perinephritis, inflammation of the capsule, and paranephritis, inflammation of the fat tissue about it, but one could hardly exist without the other and they do not need separate description. The collection of pus may be relatively large and compress the kidney, causing atrophy, or setting up suppuration in it; or it may burrow along the posterior abdominal wall and reach the pelvis, pointing under Poupart's ligament, in the vagina or finding a vent elsewhere. A chronic fibrous hyperplasia may convert the perirenal tissue into a dense mass of scar formation, and either this or the purulent form may arise in connection with chronic suppurative lesions of the kidney.

Ureter.

Malformations of the ureter may be found as entire absence of one, common with horseshoe kidney, or one or both may be double for a part of its length or the whole of it; partial doubling commonly affects the upper end. The lower ends may blend and open by one orifice in the bladder or unusual terminations may occur, as in the vagina or the urethra.

With congenital or acquired stenosis of a ureter and preservation of the upper lumen, the part above the narrowing may dilate. The obstacle may be as far away as the prostate, which prevents complete emptying of the bladder when hypertrophied and thus causes urine to accumulate in the ureter and dilate its whole extent; or inflammatory adhesions or calculi may close the upper end and only the pelvis of the kidney dilates. When there is no inflammation in the pelvis the condition is called *hydronephrosis* and the retention of urine may be so great as gradually to destroy the kidney by pressure atrophy. Similar results may be caused in floating kidney, the organ falling enough to produce a sharp bend in the ureter which prevents the passage of urine. Outside pressure, as from tumors, is less often the cause.

Hyperemia in the ureter may follow the passage of mechanical or chemical irritants, calculi, poisons and parasites; the urine may contain blood from this cause.

Inflammation of the ureter, especially important in the pelvis of the kidney, arises from similar causes, extension of inflammation upward, alkaline decomposition of the urine and especially irritation by calculi. The inflammation in mild cases is a simple catarrhal process and may leave no effects; with pyogenic organisms the mucous membrane is more swollen and hyperemic and small submucous hemorrhages may occur. The exudate is purulent and mixed with desquamated epithelial cells, and in some cases ulceration and necrosis of the mucous surface may follow (diphtheritic pyelitis). Such a purulent pyelitis almost invariably attacks the kidney tissue, causing acute interstitial (suppurative) nephritis.

Chronic pyelitis causes a thickened mucous membrane with shallow ulcerations, in which there may be a deposit of mineral salts derived from the stagnant urine, and often the contents of the dilated pelvis is composed of a mixture of pus, shreds of mucous membrane, urinary concretions and decomposed urine. The kidney may be the seat of chronic nephritis or suppuration or of atrophy from the pressure, and these conditions are at times combined.

Tuberculosis of the renal pelvis, pyelitis tuberculosa, usually complicates similar lesions in the testicles, seminal vesicles or bladder. One or both sides may be involved. The disease is chronic in its course, and beside the lesions mentioned above, presents tubercular infiltration and softening of the walls. The process usually extends to the kidney also.

Tumors.—Small cysts and polypoid growths are not uncommon in the ureter ; other tumors are almost always secondary to those of the kidney.

Parasites may pass through the ureter from the kidney or remain there for a time; as the distomum hematobium in chyuria, round worms from fistulous communication with the intestine and echinococcus from rupture into the tube.

Bladder.

Malformations.—The most important is the result of imperfect closure of the parts in the anterior median line over the bladder, by which it lies exposed. It occurs most often in males. The organ may be apparently normal but usually its anterior wall is lacking and the congested prolapsed posterior wall lies in the hypogastrium ; fissure of the urethra commonly coexists (epispadias). In the most severe form the intestine opens on the posterior wall of the bladder. Other uncommon conditions are partial or complete division of the organ by an antero-posterior septum and absence of the viscus, the ureters opening into the urethra directly.

As an acquired condition the bladder may be much dilated by chronic obstacles to urination and accumulation of the fluid, when the wall of the organ is apt to show marked hypertrophy of its muscular fibers with sacculated protrusions between them. Acute distention results in thinning of the wall and rupture is possible, either with or without injury such as falls and contusions.

In dislocations of the uterus, destruction of the perineal body and great dilatation of the urethra in women, the bladder may be inverted through the vagina, making *cystocele*, or prolapse through the meatus.

Wounds of the bladder may result from cutting or piercing instruments, rupture when distended by jumping from a high place and alighting solidly on the heels or buttocks, contusions with fracture of the pelvic bones, pressure of the head in parturition with necrosis of the injured part. Fistulæ may be the result of ulceration in the bladder, softening of tumors, and abscess formation in the adjacent tissues. Fistulæ may connect two mucous surfaces, as bladder and vagina (bi-mucous), or open externally, or burrow into the perineum or the thigh.

Hyperemia follows irritation by substances in the urine, calculi in the bladder, and accompanies inflammation of the organ itself and of other parts (puerperal metritis). It may lead to acute catarrhal inflammation. *Passive congestion* is the result of obstructed venous return, general or local, and causes varicose dilatation of the vesical veins and chronic cystitis. A severe form of hyperemia results from too rapid emptying of a bladder which for long periods has held large amounts of residual urine, as in the prostatic hypertrophies of old men; it may lead to hemorrhage and fatal syncope.

Other causes of hemorrhage are wounds, general diseases like purpura, ulceration, and tumors, especially if malignant.

Inflammation. *Acute Catarrhal Cystitis.*—Mechanical irritation by stones and foreign bodies, chemical irritation by decomposing urine or substances excreted in it (cantharides), extension upward from the urethra in acute and chronic inflammation or through the bladder wall in prostatitis, and organisms introduced in careless use of instruments, are the commonest causes of simple catarrhal inflammation of the bladder.

During life the mucous membrane is actively hyperemic; after death this may have disappeared. The bladder may be found nearly empty, corresponding to the irritability of the viscus during life, but if it contains urine there is usually a copious sediment of desquamated epithelium, mucus, pus-cells and crystals. The lesions may end in complete recovery or persist in a subacute or chronic form.

In *chronic cystitis* the mucous membrane, as in other places, becomes much thickened, polypoid growths form, there is constant and profuse production of mucus and desquamated cells, hemorrhages may occur and where they remain beneath the mucous surface the result may be deep pigmentation. Following these changes shallow ulcers often develop, in the lower segment of the bladder usually, and deposits of lime and other salts in the necrotic tissue occur. The muscular fibers hypertrophy most markedly when there is some obstruction to the flow of urine, such as prostatic increase or urethral stricture. The course of the disease may begin with such an obstruction, muscular hypertrophy follows which after a time loses its compensatory power, the urine accumulates and sooner or later undergoes fermentation from the entrance of organisms; cystitis follows at once and converts the case from a simple to a very dangerous disease. Injuries to the spinal cord cause not only paralysis of the muscular fibers of the bladder, with accumulation of urine, but the nutrition of its wall suffers so severely that the irritant urine causes rapid necrosis of the mucous membrane; to this condition the name diphtheritic cystitis has been given. Perforation into the peritoneum or the tissues of the pelvis may follow, with rapidly fatal peritonitis or gangrene of the soft parts.

Phlegmonous cystitis arises from extension of superficial ulcers of the mucous surface deeper into the wall, with suppurative inflammation; this may perforate into the pelvic tissues and cause phlegmonous paracystitis and involve the other pelvic organs.

The organism most commonly found in cystitis, beside the micrococcus ureæ which produces fermentation of the urine, is the b. coli communis which has so marked a pyogenic influence out of its proper habitat; less often the gonococcus, b. proteus and streptococci are the forms encountered.

Tuberculosis.—Occasionally tubercles develop in the bladder with pulmonary and intestinal disease, but most often the lesion is associated with invasion of the testicle (epididymis), seminal vesicle or prostate. Some cases appear to result from extension downward of renal tuberculosis but in the majority of cases the disease progresses in the other direction. The tubercles are usually found in the trigone and spreading from here a large part of the lower half of the bladder may present the miliary forms or caseous and ulcerating lesions. Cystitis of chronic type always accompanies the disease and the mucous membrane may show ulcers which are not tubercular, sometimes partly covered with false membrane, as well as those which are clearly caseous. Deposit of urinary salts is common in such ulcers. The urine may be either alkaline or acid in reaction, and blood, albumin, pus-cells and tubercle bacilli are commonly found in it. The disease is almost unknown except in males, corresponding to the greater frequency of tuberculosis of the other genito-urinary organs in this sex.

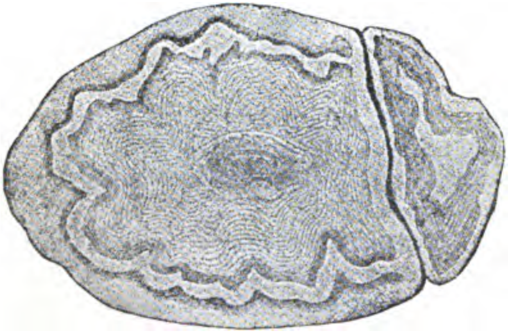
Foreign bodies are more common in the bladders of hysterical women than in the male bladder, and the list of such materials introduced includes nearly everything which may be passed into the urethra.

Calculi.—The bladder may contain from one to hundreds of calculi, the more numerous being usually small, the single ones reaching a size of several cm. in diameter and a weight of 1,000 gm., and while either sex at any age may suffer from the disease, it is most common in men in middle life or after. The stones may lie free in the bladder, and with several present opposed surfaces are often slowly ground into polished facets, or they may lie in diverticula between the separated fibers of the wall.

The form of the single stone is most commonly ovoid, seldom spherical; the surface may be fairly smooth or covered by small projections (mulberry form); the consistence varies from the hard urate calculus to softer masses of cystine, which if pure are wax-like; the color is blackish, gray or pale yellow; on section it is the rule to find concentric lines showing successive deposits, and a nucleus about which the process has started. Calculi of only one constituent are unusual, most often several varieties of mineral matter are found; often a stone of one kind (uric acid and urates) will be the nucleus for the deposit of lime and other salts.

The process consists of alternate coagulation of organic matter about a nucleus and deposit in this of urinary mineral matters; the organic matter acts as a stroma to hold the inorganic elements together, and the latter may be dissolved out and leave the organic framework or the albuminous matter may be removed by digestion and leave the minerals. The nucleus may be a foreign body, as a bit of a catheter, a blood clot or a lump of inspissated mucus or albuminous matter, and a minute stone thus started may in turn become the nucleus for a different formation; but the various layers are always cemented together by coagulated albuminous matter and it is not uncommon to find a large stone covered by such material in which little spots of new deposit are forming. The stone may begin to form in the renal pelvis (uric acid) and continue to grow after it reaches the bladder.

FIG. 26.



SECTION OF TWO VESICAL CALCULI. (*Ziegler.*)

The results of vesical calculi are partly mechanical, injury to the wall by jolting, occlusion of the meatus internus so that urination is possible only when the patient is prone, and partly the result of inflammation, for cystitis is the rule with calculus.

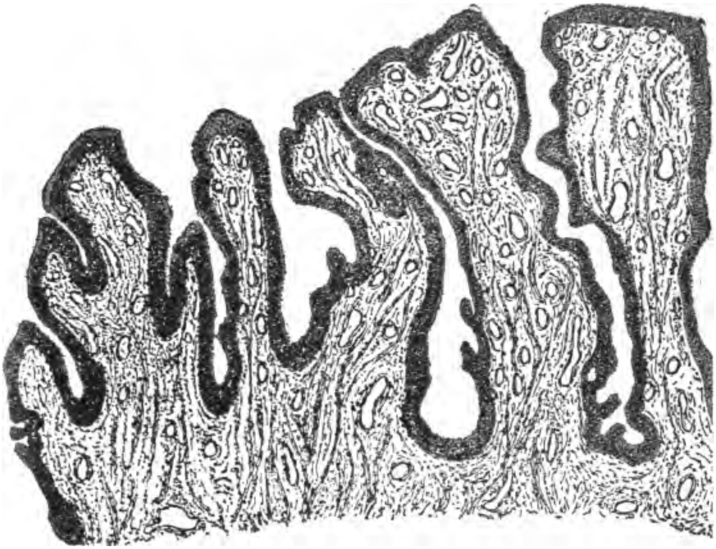
Straining at urination with hypertrophy of the detrusor fibers, pain, tenesmus, bloody urine, sudden stoppage of the stream, decomposition of urine and ammoniacal poisoning, residual urine and retention are the common results of the condition; to these must be added the pain and injury due to passage of small fragments through the urethra. Rarely the bladder wall is perforated by ulceration and the stone lodges in the pelvic tissues or passes into the vagina or rectum.

Varieties of vesical calculus (according to the material most largely present).

1. Uric acid; small, roundish, hard, smooth or granular, yellow, red or brown, concentric layers.
2. Urates; salts of ammonia and magnesia, often surrounded by phosphates, small, roundish or flattened, grayish or pale yellow, smooth or granular.
3. Phosphate of calcium; small or medium sized, rough, sandy surface, white with a gray or yellow tinge, hard, concentrically marked.
4. Triple phosphates; large, rough surface, grayish.
5. Calcium carbonate; small, white, friable.
6. Calcium oxalate; may be large, hard, brown, heavy, surface nodular.
7. Cystin; medium size, yellow brown, soft, round or ovoid.
8. Xanthin; small, ovoid, reddish-brown; these last two forms are among the unusual varieties; they may be the nuclei for phosphatic and uratic calculi.

Tumors.—Polypoid growths have been mentioned in connection with chronic inflammation; they are usually small. Fibroma occurs as a papillary tumor in the lower portions of the bladder and is very vascular, hence hemorrhages are common and may produce fatal anemia, bits of the tumor may separate and block the internal meatus, and chronic cystitis is commonly associated with the lesion. This tumor is a good example of a benign form with malignant consequences because of the accident of location. Carcinoma may develop upon such a basis or be secondary to carcinoma of the rectum, cervix and vagina, prostate, testicle and other organs.

FIG. 27.



PAPILLOMA OF THE BLADDER. (*Delafield & Prudden.*)
Fibro-cellular tissue, rich in vessels, invested with irregular layers of epithelium.

Parasites in the bladder occur as accidental results of fistulæ between it and the gut, or wandering oxyuris from the rectum, or rupture of echinococcus cysts into the ureters; filaria are abundant in chyluria.

Urethra.

Malformations are commonly imperfect closure of the tube, epispadias and hypospadias, non-development of a part, causing atresia urethræ, and variations in the passage of the tube through the prostate gland.

Injuries may be the result of external violence, as falling astride a narrow object, or internal traumatism, as attempts to introduce instruments, or, in women, pressure of the child's head during parturition, use of forceps, etc. Such injuries result in hemorrhage and extravasation of urine, and the tissues infiltrated are apt to suffer phlegmonous or gangrenous lesions with great rapidity. This is commonest in the male and may reach an extent which includes the lower anterior abdominal wall and the upper part of the thigh as well as the perineal tissues. This course of extravasated urine depends upon the union posteriorly of the deep perineal fascia with the deep layer of the superficial fascia, " false passages " from the urethra usually opening into the space between. Fistulæ opening externally or into the ischio-rectal and other spaces may remain.

Inflammation.—*Urethritis* in the vast majority of cases is a specific disease produced by contact with infected persons, the bacterial agent being the gonococcus. Simple catarrhal urethritis may occur, as the result of the passage of calculi, irritating drugs, and the presence of pyogenic organisms. Gonorrheal inflammation may be limited to the anterior portion of the urethra or travel through its whole extent and invade the bladder, prostatic glands, seminal vesicles and vas deferens; and in women, bladder, vagina, uterus and tubes are often involved. In the fallopian tubes such an inflammation may become chronic and distend the parts with slow accumulation of pus. The inflammation is exudative, the epithelial cells desquamate in great numbers, pus-cells abound.

In the bodies of the cells, both epithelial and pus, at times in their nuclei, as well as free in the tissue and the discharge, the characteristic diplococci are found. They lie in twos and fours or irregular groups and are arranged with their flat or slightly concave surfaces opposed, with a narrow space between. They stain easily with anilin dyes and are decolorized with Gram's fluid; this fact and the difficulty of cultivating them are important characteristics. Pus organisms occur at first, but as the disease becomes chronic all forms of bacteria tend to disappear from the discharge, which becomes thin and colorless and lessens in amount. But in the crypts of the mucous glands along the urethra the gonococcus may linger for long periods, not very injurious for the patient but able to convey the infection to another.

Other parts attacked are the rectum, the conjunctiva and at times the joints (gonorrheal rheumatism); the diplococcus has been recovered from these places as well as from tendon sheaths, the glands of Bartholin and peritonitic pus.

Chronic urethritis frequently follows the acute disease and the part most often involved is the posterior urethra; it spreads from here to the prostate and seminal vesicles in many cases. The discharge is thin, perhaps limited to a drop or two of glairy mucus noticed only in the morning; the part affected becomes covered with little papillæ which at times are sensitive and bleed easily. A more serious sequel of urethritis is the formation of fibrous thickenings in the wall of the tube, either along the floor or in ring form. Such *strictures* probably begin as productive fibrosis about a small ulcer or a suppurating mucous gland most commonly in the membranous portion, and the tendency is toward contraction of the tissue and stenosis or closure of the lumen; such a mechanical obstacle produces hypertrophy of the bladder, dilatation of this viscus, the ureter and the renal pelvis, with decomposition of stagnant urine, or acute retention. Other complications are inflammation of the epididymis and the testicle.

Tumors are not common in the urethra except when it is secondarily involved. Condylomata are observed about the meatus in the female, polypoid growths occur in either sex, carcinoma occurs with similar disease of the glans penis, the prostate, and the cervix uteri; but the urethra may escape even with extensive lesions of the neighboring parts.

Tuberculosis may invade the urethra as lupus from the skin or when the kidney and bladder are diseased.

Syphilis may occur as a chancre in or about the outer portion of the urethra.

CHAPTER VII.

REPRODUCTIVE SYSTEM.

Male Organs.

Malformations.—The penis may be absent or rudimentary, or in rare cases double; the prepuce may be absent, the urethra may open on the dorsum of the penis (*epispadias*) or on the under side (*hypospadias*); most cases of hermaphrodism are only severe degrees of such imperfect closure. The testicles may fail to descend into the scrotum (*cryptorchism*), and then be incapable of functionating; the inner genitals seldom present malformations.

Injuries of Penis and Scrotum.—Wounds of the penis cause hemorrhage, more marked when occurring during erection, and if the urethra is involved fistulæ or extravasation of urine may result. *Ganglion* of the penis is the name given to hard fibrous masses in the corpora cavernosa after injury with hemorrhage. Rupture or fracture of the penis may be due to violence during erection, the fibrous sheath of the organ is torn and severe bleeding follows. The penis may also suffer luxation, being crowded up under the abdominal integument by severe pressure, as of a wagon wheel. Permanent bending or twisting of the organ may result.

Inflammation of the glans is called *balanitis*, of the prepuce, *posthitis;* both usually come from lack of cleanliness or complicate *phimosis*, inability to retract the prepuce; *paraphimosis* results when a tight prepuce can not be drawn over the glans again after retraction, owing to edema of the tissue. Inflammation of the deeper structures of the penis seldom occurs.

Ulcers.—The commonest are chancre, the initial lesion of syphilis, and chancroids, and their usual seat is on the glans, behind the corona or elsewhere. Lupus may produce an indolent ulcer of the glans, carcinoma of the part often ulcerates, condylomata at times affect the glans, prepuce and scrotum.

Tumors.—Beside venereal warts, epithelioma affects the glans penis and the prepuce; it may be papillary and in any case ulcerates rapidly. It is commonest in late adult life.

Scrotal epithelioma (chimney sweeper's cancer) occurs as flat masses of induration in the skin with a tendency to ulcerate; it extends directly to the penis and through the lymphatics to the inguinal nodes. It affects workers in paraffin also.

In oriental countries the scrotum is at times the seat of elephantiasis, enormously enlarged, hanging nearly to the knees of the patient, the penis retracted within the leathery tissues leaves a funnel-shaped opening through which the urine discharges; fissures and ulcers are common in the swollen skin. In some cases the lymph spaces of the part are dilated and contain numbers of filaria.

Testes.

Hydrocele, or dropsy of the tunica vaginalis, may occur with general edema, or, when the way to the peritoneum is open, ascitic fluid fills the serous sac of the scrotum also; a hydrocele of the cord also occurs. Injuries to the testis are sometimes followed by serous collections in the sac; epididymitis may be complicated by the same. In distinction to these acute cases of hydrocele, where the fluid rapidly accumulates and may be serous or mixed with blood and pus, chronic hydrocele grows slowly, the fluid is clear, and large amounts may collect and return after tapping. Papillary outgrowths from the epididymis (*periorchitis prolifera*) and testis may develop, and these may separate after lime salts are deposited in them and lie as free bodies in the cavity. Filaria cause a kind of hydrocele in which the fluid is milky (*galactocele*).

Spermatocele is the name given to a cystic tumor containing fluid and spermatozoa which develops near the head of the epididymis.

Hematocele is either a hemorrhage into the tissues of the scrotum (hematoma) or a collection of blood within the vaginal sac, as in scurvy or with acute hydrocele. Injuries usually cause extra-vaginal hematoma.

Embolism of the spermatic artery and thrombosis of the pampiniform plexus in rare cases lead to a rapid spontaneous gangrene of the testicle; interference with the vessels of the cord may lead to fatty degeneration or necrosis of the organ.

Inflammation.—*Orchitis* may be the result of injury to the testis or complicate gonorrhea; when the disease is limited to the epididymis it is called *epididymitis*, when the serous coat is especially involved it is called *periorchitis*. With small-pox and some other diseases, and in a "metastatic" form with mumps, inflammation of the testis is occasionally seen. The acute form is attended with great swelling, and owing to the firm tunica albuginea the part is hard and very painful. The disease may end in complete restoration, or suppuration may occur, and when the tunica albuginea is broken through a fungous condition of the testis results.

Chronic orchitis may follow the acute form or complicate syphilis. The inflammation is chiefly productive and reduces the size and destroys the function of the organ.

Atrophy of the testis occurs with loss of function and after operative procedures involving the spermatic cord.

Tumors.—The commonest benign neoplasms of the testis are fibroma, lipoma and myoma; chondroma occurs simply or combined with sarcoma. Sarcoma occurs in all its forms in the testicle and the same tumor may present a different constitution in various parts, as also in the parotid gland. Carcinoma is usually of the encephaloid type and combinations with adenoma and chondroma have been described. Metastases are usually rapid in all the malignant forms of tumor, as well as extension to surrounding parts by direct invasion.

Tuberculosis often begins in the canals of the epididymis and from here affects the testis later; it is apt to invade the seminal vesicles and the prostate also, and from here may reach the kidney by way of the urinary tract or the lymphatics. The disease develops slowly and the caseous masses may reach a large size; fistulæ opening externally may form and discharge pus and broken-down material. The disease is usually secondary to pulmonary tuberculosis, though this may be clinically less serious than the genital disease, and the question of transmission during intercourse naturally suggests itself, especially during the early stages.

Leukemic and lepra nodules have been found in the testis but are not of pathological importance.

Seminal Vesicles.

Acute *inflammation*, usually complicating gonorrhea, may be accompanied by imperfect erection, premature ejaculation, bloody semen and pain during and after coitus. Chronic inflammation is rather common as the result of posterior urethritis and often causes nervous symptoms, neurasthenia and melancholy. The vesicles can be felt by rectal touch as swollen, tender and thickened cords, usually distended with fluid; on massage this is pressed into the urethra and passed with the urine. Under the microscope hyaline crescentic bodies, dead or imperfect spermatozoa and Charcot crystals may be found, or in many cases complete aspermia. The dilatation is due to inflammatory products about the entrance of the ducts into the prostatic tissue.

Tuberculosis complicating pulmonary disease frequently starts in the vesicles, the bacilli being excreted with the semen and attacking the vesicles while stored in them; from here the disease may spread to the other pelvic organs, or caseous abscesses may form and be discharged by the urethra and apparent recovery follow.

Primary tumors are very infrequent. Small retention cysts rather common. Small concretions form at times from coagulated contents in which salts are deposited.

Prostate.

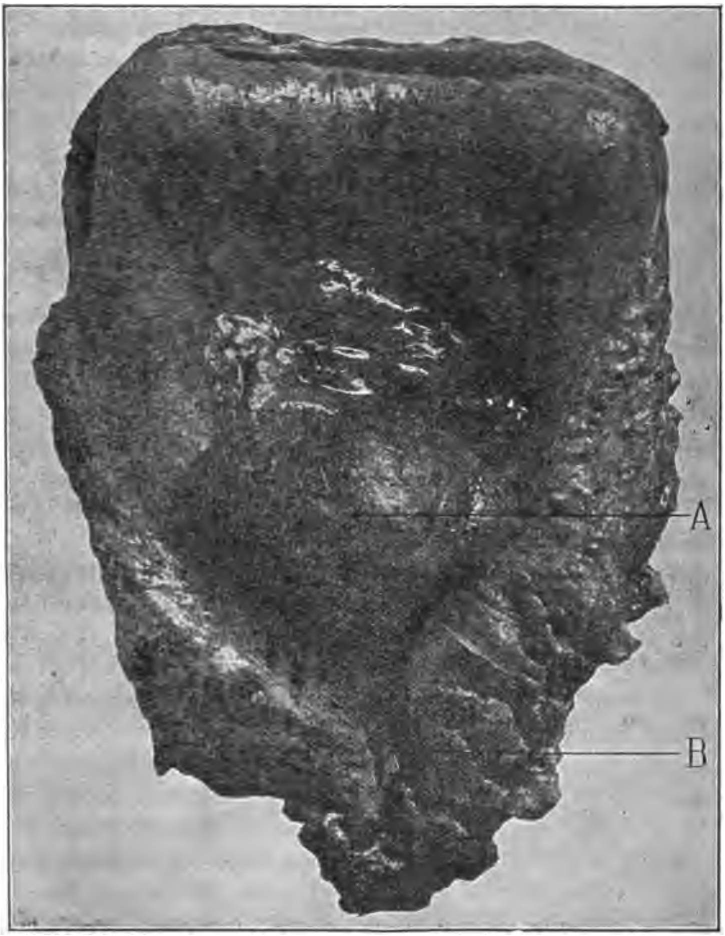
Acute *inflammation* of the gland may complicate gonorrhea, and, from the rapid swelling induced, retention of urine may follow. The exudate is chiefly serous and may be rapidly absorbed. From this form a suppurative inflammation of more chronic character may develop, resulting in abscess in one of the lobes. Injury to the prostatic urethra and suppuration in the pelvic tissue may also cause abscess of the gland; in pyemia emboli of pyogenic organisms produce numerous small foci of pus. As a rule prostatic abscess empties into the urethra; less often the limits of the capsule are passed and the pus burrows into the rectum or along it to the perineal surface. When the urethra is opened extravasation of urine into the pelvic tissues occurs. Phlebitis may occur in the prostatic plexus, and, if septic, emboli from here may produce general infection. The gonococcus may gain an entrance to the circulation in a similar way and cause malignant endocarditis.

Concretions in old age are very common in the prostate. They may be visible to the naked eye as small brownish granules scattered over the section. Under the microscope the older bodies are striated, concentrically and in radii, the rings corresponding to increases in size and the radii to drying. They stain with iodine, either blue or reddish-brown, or not at all. Deposits of lime salts often occur about these bodies.

Atrophy of the gland is a regular senile change, follows castration (at times), and results from fibrous contraction with degeneration of the glandular epithelium.

Hypertrophy occurs in middle age and later, and because of the mechanical conditions is of great clinical and pathological importance. Either the stroma or the glandular tissue may be involved and the three common forms arise as one of these predominates or the two co-exist. Thus the hypertrophied organ may be small and hard, chiefly made up of fibrous tissue and

FIG. 28.



MIDDLE LOBE HYPERTROPHY. (*Stengel.*)

A, hypertrophied middle lobe; *B*, urethra. The muscular tissue of the wall is much hypertrophied.

muscular bundles, the glands compressed and their epithelium atrophic; or it may be large and soft, the glands dilated, often containing concretions, muscular fibers increased in size and number; or both pathological changes may exist.

The gland normally has but two lobes; when hypertrophied it may have four. Either lateral lobe or both may grow, or the posterior isthmus below the internal meatus may be the chief seat of the hypertrophy, rising like a round knob into the bladder and transformed into a "middle lobe" only as the result of a pathological process; a similar smaller isthmus of gland tissue, not always present, which arches over the meatus, may constitute a fourth imperfect (pathological) lobe.

With middle lobe hypertrophy the new growth acts as a ball valve and prevents expulsion of urine, increased pressure by the vesical and abdominal muscles only pressing the tumor more tightly against the meatus internus. One-sided hypertrophy distorts the prostatic urethra, when bilateral the passage is much narrowed; in any case the prostatic urethra is lengthened and more curved than normal. The results for the bladder are mechanical at first, with hypertrophy of the muscular trabeculæ, and at times sacculation between them; later, when residual urine accumulates and decomposes, and the compensatory power of the bladder is lost, the case becomes graver, fever, sweating and other general symptoms are added.

The gross appearance of the organ varies with the kind of hypertrophy, and its seat; the cut surface may be soft and pale in the large form or harder when chiefly fibrous; here and there may be areas with concentric arrangement of fibers like vortices separated by interlacing bands, as if the gland were made up of many smaller ones tightly pressed together. The capsula propria and the investment derived from the recto-vesical fascia may have become blended so that they cannot be separated, with only traces of the plexus of veins normally between them; out of this thickened capsule the hypertrophied gland may at times be shelled with slight effort. Microscopically the appearances respond to the tissue which is most abundant.

The cause of prostatic hypertrophy is probably long-continued congestion, kept up for years; this may be the result of masturbation, imperfect intercourse (coitus reservatus), habitual excitement of the organ without the normal relief of the functional hyperemia consequent on normal ejaculation, extension of chronic inflammation from the posterior urethra, and varicose conditions of the veins with impeded return.

Tumors.—Both sarcoma and carcinoma are very rare in the prostate, either as primary or as secondary tumors. Tuberculosis almost always occurs with other genito-urinary forms of the disease and requires no special discussion.

Cowper's Gland.

In the course of gonorrhea the gland may become inflamed and suppurate; the pus may burrow and leave a fistula communicating with the urethra. When the duct is stenosed retention cyst occurs, and with chronic inflammation fibrous induration is the common result.

Female Organs.

Vulva.

Malformations are not of great pathological importance with the exception of atresia, partial or complete, and unusual forms of the hymen which may have a legal interest; this membrane may be absent, or fimbriated, or a narrow ring of tissue with a wide lumen, or imperforate.

Injuries.—The most important are those received during parturition. These are chiefly pressure effects, which may produce sloughing of the labia, hematoma in one or both, perhaps extending into the vagina, and tears of the posterior commissure which may extend through the entire perineal body into the rectum. Slight tears usually heal rapidly in the absence of infection. Contusions may cause inflammation of the labia and Bartholin's gland with abscess formation.

Hyperemia may be passive and accompanied with edema in pregnancy and hydremic conditions. Acute congestion occurs with various skin eruptions and with more extensive inflammation. Acute vulvitis may follow injury during attempted coitus, irritation from scratching (with pruritus), and in children thread worms leave the rectum, especially at night, and irritate the parts. The usual symptoms of redness, swelling and increased secretion follow. Gonorrhea may produce such an external condition and often the gland of Bartholin on either side is also involved and suppurates. Diphtheria and phlegmonous inflammation occur at times, the last may result from pus burrowing from distant parts.

Ulcers on the vulva may be found with the above lesions, and in syphilitic patients both chancroids, often multiple, and chancres occur here. Gangrene may follow severe injury or in the cachectic occur spontaneously; a line of demarcation may form and the process heal with large contracting scars, but the probable course is to a fatal ending.

Tumors.—Soft fibroma and myoma sometimes occur on the labium majus or about the urethral opening, and reach a large size, usually pedicled and covered by thinned non-adherent skin. Lipoma and chondroma have been observed. Papillomata are far more common, in the form of broad or acuminate condyloma, with syphilis; a non-specific form, of small size but extremely sensitive, develops in the meatus and is known as urethral caruncle. Carcinoma is rare but may develop from the clitoris, the labium minus or the epithelium of Bartholin's gland. It may extend in any direction and soon breaks down and ulcerates.

Tuberculosis occurs as lupus, with slow progress by ulceration while older parts cicatrize imperfectly.

Vagina.

Malformation.—Atresia may occur at the hymen or elsewhere in the canal; it may not be discovered till menstruation begins and the monthly discharge distends the canal (*hematocolpos*). The vagina may be imperforate for its whole length, only a fibrous cord occupying its main axis, or it may be of normal size but divided by a septum; in the latter case the uterus is also double as a rule.

Dislocation of the canal usually follows rupture of the perineum or uterine displacements or both combined. Slight degrees of vaginal prolapse may arise from looseness of the attachments of either wall; the anterior wall is more often involved and may drag down the uterus with it. When this attains a marked development the bladder follows and the condition is known as *vaginal cystocele;* the name *phantom tumor* is also used, for the apparent tumor is easily replaced on change of position or by the examining finger. When the posterior wall and the anterior rectal prolapse it is called *vaginal rectocele.* With complete prolapse of the uterus both walls may be dragged out and their surface in time become dry and leathery, resembling epidermis.

Wounds of the vagina may arise from foreign bodies, violent coitus with great disparity of the parts, injuries during parturition from the head or obstetric instruments. Perineal rupture if complete also involves the vagina. Slight wounds often heal quickly unless infected; then suppuration may occur and reach the deeper tissue, and the scars may make a partial or a complete stricture of the canal. Another result of injuries is the formation of *fistulæ,* which may join bladder, vagina and rectum and involve the uterus and the pelvic tissues also. The lumen of the fistula may be very fine or large enough to permit the passage of urine and feces; severe inflammation and even gangrene may result; spontaneous healing is seldom secured.

Inflammation.—Acute *vaginitis* (or *colpitis*) may be a mild and temporary catarrhal inflammation, due to hot douches, mechanical irritation and, in children especially, the presence of oxyuris. The severe attacks may usually be referred to gonorrheal infection. The mucous membrane is then swollen, hyperemic, covered with a muco-purulent discharge, and the urethra usually shares in the process; shallow ulceration is common. A destructive vaginitis with necrosis of the epithelium and formation of a pseudo-membrane occurs in cachectic patients with pyemia, variola and other infectious diseases; it may accompany true diphtheria of the pharynx but commonly is quite distinct. The lesion ends in recovery with large scar formation and contraction, or in phlegmonous and gangrenous destruction of the tissues.

Chronic catarrhal vaginitis is usually known from the copious discharge as fluor albus or "the whites." The discharge is yellow or creamy, thick and purulent, or thinner and acrid, acid in reaction, at times greenish; the cervical canal is commonly involved also. In other cases the mucous membrane is dry and thickened with almost no excretion.

Tumors.—Fibroma develops in the vagina as polypoid tumors of fibrous or fibro-myomatous type and also as acuminate condylomata. Sarcoma is uncommon. Carcinoma may occur as flat or papillary epithelioma, commonly in the upper portion of the canal, and at times develops into a large cauliflower mass; secondarily the vagina is usually involved with uterine cancer of the cervical region and with similar disease of the anterior rectal wall.

Tuberculosis may appear either as lupus or as scattered ulcers, the former extending from the skin and the latter from the uterus. Syphilitic ulcers of all varieties may be found in the vagina.

Uterus.

Malformation.—Imperfect development of Müller's ducts results in absent or rudimentary uterus or vagina, or both; a very small uterus is termed infantile or fetal and is remarkable for the disproportion between body and cervix, the latter forming most of the organ; the ovaries also may be lacking or undeveloped. The adjacent walls of the Müllerian ducts, which disappear normally leaving but one cavity in the genital canal, may persist more or less completely and give rise to the bicorn and bipartite uterus and the septum may divide the vagina also into two canals. Atresia of the cervix may lead to accumulation of menstrual blood (hematometra), or of muco-serum (hydrometra); inflammation and suppuration may follow.

Displacements.—The form and anatomical relations of the uterus both permit various displacements and make their results serious. When the organ moves as a whole, without alteration in its proper form, it is called *version*, of which the most important are anteversion, retroversion and lateral version. The effects are chiefly mechanical, from pressure on bladder and rectum or dragging on ligaments, and are less severe than in flexion. When the uterus suffers a bending in itself the condition is called *flexion*, and the degrees correspond to those of version, ante-, retro- and lateral flexion. The results are chiefly due to consequent narrowing of the uterine lumen, difficult menstruation and retained flow. The condition may be caused by pressure from above, as of the clothes, contraction of inflammatory adhesions and other external influences, and in an imperfectly restored uterus after childbirth (subinvolution), loss of tone in its walls and weight and congestion of the fundus may precede the condition. Entire movement of the organ upward has but little importance; the contrary condition, *prolapsus uteri*, is observed when the inferior support is lessened and the vulva patulous, as after rupture of the perineum. Three grades of this condition are distinguished, simple descent, the uterus not leaving the pelvis; partial prolapse, the organ being out of the pelvis but not entirely; complete prolapse, the uterus, and with it the vaginal walls and inside them the vesical and rectal walls, protruding from the vulva.

Inversion of the uterus occurs after labor or with submucous polyps of large size, the traction turning it inside out more or less completely; this may be associated with prolapse.

Rupture of the uterus may result from accumulation of fluid within it, and thinning and necrosis of its wall, but this is very unusual. The accident more often takes place in the pregnant organ, and here there may be predisposing conditions, as malignant disease, destroying the muscular wall. Other cases result from convulsive efforts at expulsion of the fetus, disproportion between uterus and child, pregnancy in one half of a double uterus. The tear in the uterine wall is most often in the region of the cervix or just above it and proceeds from within outward; tears starting on the peritoneal surface are practically unknown. The depth of the injury may be less than the thickness of the wall (incomplete rupture), or pass entirely through it and not involve the serous surface, or include this and other parts also, as bladder and rectum; copious hemorrhage into the peritoneum may be immediately fatal and with it the fetus and its envelopes may leave the uterus and be found in the clot. In very favorable cases, with but little bleeding, peritonitis may close in the fetus, adhesions may form, and purulent softening open a way for the escape of the dead fetus through the intestine. In any case the termination is usually fatal, from shock and bleeding, peritonitis or chronic suppuration.

Tears spontaneously produced usually lie in the direction of the long axis of the uterus, in or near the cervix; if caused by version or other obstetric procedure they may be oblique, transverse, and in other parts of the organ. Slight injuries of this kind usually heal quickly in the absence of infection. Other perforations, as from a curette (and the septic uterus as after abortions is very easily perforated) usually occur in the fundus and make small linear or branched openings on the peritoneal surface with inflamed or necrotic edges, the wound widening toward the mucous surface.

Circulatory Disorders.—*Hyperemia* of the uterus occurs normally in a cycle of about twenty-eight days; death during menstruation leaves the mucous membrane swollen and congested, its upper layers degenerated or lost. Menstruation is associated with serous swelling of the mucosa and separation of its elements, round-cell increase between the glands, and increase in the length of the tubular glands; the swelling subsides rapidly with cessation of bleeding from the dilated capillaries, the upper layer of mucous membrane is exfoliated and the glands become spiral again. When the entire lining of the uterus, as a cast of its cavity, is extruded at menstruation, with pain and uterine contractions, the anomaly is known as *membranous dysmenorrhea*. The cause is usually an inflammation of the mucosa (exfoliative endometritis). The membrane is made up of uterine glands held together by very cellular connective tissue holding red cells in its meshes; in other cases the membrane consists chiefly of fibrin and blood-cells.

Certain infectious diseases, blood states and local conditions may cause uterine *hemorrhage* apart from menstruation; among these are cholera and small-pox, scurvy, and tumors. Hemorrhage in the tissues about the uterus may occur in the peritoneum (Douglas' pouch) or in the tissues, as of the broad ligament; the usual causes are menstrual flow from the tubes, rupture of extra-uterine pregnancy, rupture of a varicose vein or of newly formed vessels in adhesions, and injuries during parturition. The blood may form a large hematoma and in favorable cases is absorbed or encapsuled; in other cases inflammation occurs. The blood may escape through rectum or vagina after softening of their walls, or necrosis of the tissues may end fatally.

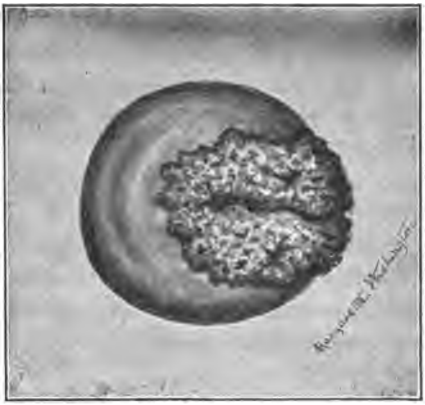
Inflammation.—The part of the organ attacked may be the serous coat (*perimetritis*), the subperitoneal tissue adjacent (*parametritis*) the wall of the organ (*metritis*) or the mucous surface (*endometritis*). In the non-pregnant uterus perimetritis is usually either acute and part of a general pelvic peritonitis, or chronic and associated with other inflammations; either may cause fibrinous exudate with adhesions or pus formation. Such adhesions are almost constant with various tumors of the uterus. Parametritis is usually an acute inflammation of the cellular pelvic tissue about the uterus and occurs with metritis and endometritis. Of these two forms many slight attacks may occur at variable periods and the pelvic contents may be firmly fastened together and to loops of intestine, the omentum, etc.

Metritis seldom occurs in the acute form except in connection with puerperal conditions. It may follow gonorrheal and other infection with marked lesions of the mucosa. Chronic metritis results from chronic congestion, subinvolution and displacements and is partly a fibrous and partly a muscular hyperplasia. The entire organ may be involved, but often the condition is limited to the inferior zone of the organ.

Acute endometritis is uncommon but may follow injury, or pyogenic and gonorrheal infection of the mucosa. It may be limited to the cervix (endocervicitis) or invade the fundus also. As a complication with infectious disease it occurs in typhoid and scarlet fevers. The mucous membrane is swollen, congested, loosened in its attachment, hemorrhagic and covered with mucopurulent exudate; severe necrosis of the mucosa gives rise to a diphtheritic form.

Chronic endometritis follows the acute disease or results from anemic and chlorotic malnutrition of the part or irritation by polypoid and other tumors. The mucous membrane is thickened and congested, its glands are hyperplastic and irregularly dilated and the stroma may also show hyperplastic changes; with the progress of the disease an atrophic stage is reached when the glands disappear and the stroma becomes indurated. This process in the cervix results in retention cysts of the glands, forming the ovules of Naboth. The discharge of chronic catarrhal endometritis may be purulent, at times mixed with blood, but in many cases it is thin and colorless. The inflammation may extend into the tubes, atresia of the cervix may result from it, and membranous dysmenorrhea is another commonly associated condition. Ulcers in the fundus sometimes accompany the process; on the cervix they are extremely common and are known as erosions. The granulation tissue may be so exuberant as to close the cervical canal. Such erosions are common after laceration of the part. More superficial lesions occur from loss of the squamous epithelium of the part; its place may be taken by cylindrical cells.

FIG. 29.



LACERATION AND EROSION OF THE CERVIX. (*Stengel.*)

Puerperal Metritis.—The recently emptied uterus presents a natural wound; this and other traumatism of any part of the parturient canal, even very shallow tears in the mucous membrane of the lower vagina, may be the entering point for the infecting organisms. Deaths from childbed fever have steadily declined in some hospitals when the nurses have been forbidden to make digital examinations of the vagina before and during labor; further decrease has followed extension of the order to the visiting students, and when nobody was excepted the mortality for long periods has been nil: such facts, and the frequency of the infection in the practice of certain physicians and midwives, suggest the source of the infection and the difficulty of avoiding it altogether, even with care. The organisms found have been strepto- and staphylococci, alone or together, the gonococcus, b. coli and b. tetani. As a rule no part of the organ escapes, and the pelvic tissues may also be inflamed; the general condition may be that of septicemia, but local septic phlebitis often results in embolic abscess in various organs (pyemic form). The veins involved may be the sinuses at the placental attachment, and when the disease arises in consequence of putrefaction of retained placenta this is the natural source of general infection. A phlegmonous form occurs with lymphangitis of the organ, abscesses forming in the wall of the fundus at the attachment of the tubes, or in the cervix, in the broad ligament, and the cellular spaces of the pelvis. Suppurative peritonitis develops early and is usually fatal.

Atrophy of the entire uterus occurs as a senile change, with obliterating arteritis of the vessels of the part; it may occur early in life after removal of the ovaries. Hypertrophy occurs with chronic metritis. Fatty degeneration may result from poisons (phosphorus) or accompany infectious disease or pressure from tumors (intramural fibromata). Amyloid changes are very infrequent.

Tuberculosis of the uterine mucosa may arise from extension of a tubal lesion. The entire mucosa may be thickened and caseous, with large losses of substance from softening, or a few miliary tubercles may be found here and there in the membrane; the disease usually accompanies other genital tuberculosis and similar disease of the peritoneum.

Tumors.—Polypi result from localized submucous hyperplasia and hang in the uterine cavity of the vagina as small red and pedicled tumors. They may be mucous, cavernous or glandular, and are of importance from the accompanying hemorrhages.

Fibromata are the commonest tumors of the uterus. They occur under the serous or the mucous coat or in the substance of the organ, and hence are called subperitoneal, submucous or intramural. They may be single or multiple and an intramural tumor may become either subserous or submucous; the latter varieties are often provided with a pedicle. In size they vary from a few millimeters to many centimeters in diameter, though the large tumors weighing twenty kilograms and more are seldom seen at present.

The consistence of the tumor varies with the proportion of fibrous tissue, but they are often very hard, sharply circumscribed by a capsule out of which they may be lifted, and poorly supplied with blood-vessels. Degeneration is common in their middle portions and lime salts may be deposited in them on the surface or diffusely; when a pedicled subserous fibroma becomes twisted on its pedicle it may become necrotic and cause suppurative peritonitis.

Microscopically these tumors usually contain some muscular fibers, often so many that they are called fibromyomata; these cells are of large size and provided with large nuclei. An apparent concentric arrangement can be made out by which the muscular and fibrous elements are disposed about a dilated capillary. Occasionally the capillaries are so large and numerous as to form telangiectatic and cavernous myomata.

The effects of uterine fibroma comprise the results of pressure on adjacent parts, displacements of the uterus, bleeding from the congested mucosa and degeneration of their tissue. The submucous forms may simulate inversion of the uterus, and are exposed to various injuries, with the danger of hemorrhage and inflammation.

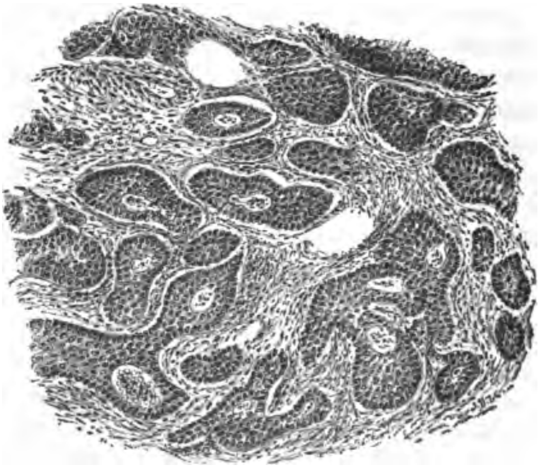
Sarcoma may develop from fibro-myomata (myoma sarcomatodes), and is commonly of the spindle-celled variety; or it may start in the deeper layers of the mucosa and form polypoid vascular tumors, commonly round-celled. Adeno-sarcoma and angio-sarcoma may develop from the endometrium. A peculiar papillary edematous sarcoma sometimes occurs in the cervix during early life. Its nature is very malignant and the microscopic structure complex, including round and stellate cells, giant cells, muscle and cartilage, in a myxomatous and edematous matrix.

Epithelial tumors of the uterus may follow the type of the squamous cells of the cervix (vaginal aspect) or the cylindrical glandular cells. It is not uncommon for these tumors to begin as a benign growth and pass over later into a true carcinoma.

Papilloma forms relatively small and hard tumors, often multiple, on the portio vaginalis; the surface of the tumor is covered by many layers of flat epithelia.

Adenoma occurs as hypertrophies of the uterine mucosa after chronic inflammation, and as polyps. A malignant form takes its origin in the mucosa of the fundus and is distinguished from the simple glandular hyperplasia by the tendency of the atypical glands to invade the muscle of the organ. In tumors of this variety the epithelium often grows with great rapidity, the acini of the glandular structure are broken through and the transition to carcinoma is complete (Adenoma destruens), Pt. I., p. 166.

FIG. 30.



CARCINOMA OF THE CERVIX. (*Stengel.*)
Cell nests in a fibrous stroma with suggestions of a lumen in the former—Adeno-carcinoma.

Carcinoma is the commonest malignant tumor of the uterus and its usual seat is the portio vaginalis. It begins on the inner surface of one of the cervical lips as an area of firm induration, not well circumscribed from the surrounding healthy tissue. The mucosa thickens and is adherent to the layers beneath.

The disease progresses till it invades the vault of the vagina and rapidly breaks down. The ureters and bladder are sooner or later involved, the rectum may be opened, and the entrance of urine and feces hastens the destructive action of the cancerous ulcerations. Metastasis to the inguinal and lumbar nodes and to the liver are common; the fundus uteri is not involved or late in the disease.

Epithelioma of the cervix differs from benign papilloma in the rapidity of its growth, the softness of its tissue and the formation of but one broadly based tumor. The growth of the new epithelial tissue occurs in two directions at once, into the muscular fibers of the cervix and out into the vagina as an increasing cauliflower growth. Both these varieties of malignant neoplasm lead to profuse hemorrhage; the epithelioma does not ulcerate or invade surrounding parts so rapidly.

Carcinoma of the body of the uterus may be of the flat-celled type, in women of advanced age, in whom the epithelium of the fundus has become squamous after chronic inflammation; otherwise the tumor is cylindrical celled, and resembles malignant adenoma in many cases.

Cysts of the uterus are rare; they arise from softening in fibro-myomata or are small retention cysts of the mucous glands, or occasionally dermoid. Cystic parasites have been encountered (echinococcus).

Ovaries and Tubes.

Either or both organs and ducts may be absent; hypoplasia is more common; occasional displacement occurs, as into a labium majus.

Acquired displacement of the ovary is common with uterine version and flexion; the ovary frequently lies in Douglas' pouch and has been found in crural and other hernias.

Congestion is normal before ovulation; passive congestion may be part of a general stasis.

Hemorrhage into the follicle follows extrusion of the ovum and forms the corpus luteum of menstruation, which soon organizes and disappears, all but a small scar on the surface of the ovary. The corpus luteum of pregnancy is larger, persists till the middle or end of pregnancy, and its luteal border is very much broader.

Inflammation accompanies ovarian tumors, extension of salpingitis and peritonitis. In the puerperal form, as in the first mentioned, one or both ovaries may be affected and the tube may also take part or escape for a time. The inflamed ovary is swollen, hyperemic and succulent or grayish-yellow in color. The process may resolve or lead to abscess formation and peritonitis. Adhesions form with surrounding viscera and remain as firm bands after the inflammation ceases.

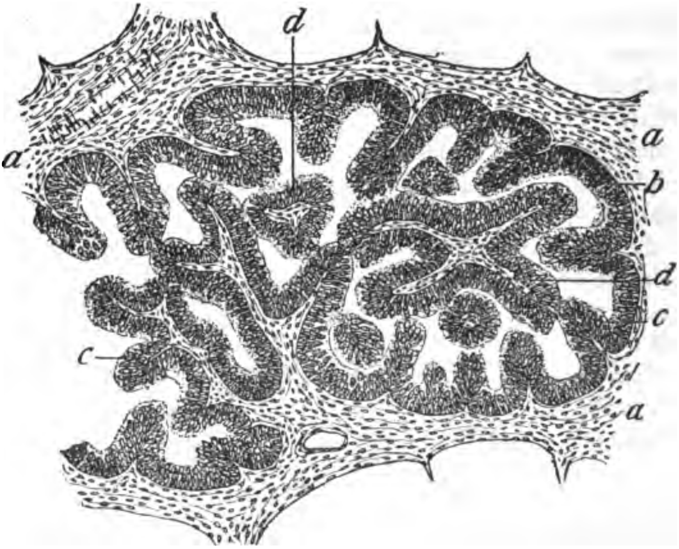
Tumors.—The most important pathological condition of the ovary is furnished by its neoplasms. They are nearly always cystic.

Fibroma occurs as simple, sometimes of large size, or as fibromyoma resembling the uterine tumors, or as fibro-sarcoma. They probably take origin in the fibrous scars left after healing of corpora lutea. Sarcoma is most often either spindle-celled or combined with fibroma; round-celled forms, angio-sarcoma and myxo-sarcoma also occur.

Cystic tumors may develop in the follicles (hydrops of the Graafian follicle) and occur singly or multiple, in the young but more frequently in older patients. They are small or of medium size, seldom much larger than the fist, and the interior is lined by a single layer of flattened epithelium. They contain clear fluid which may be thin or jelly-like, or, with hemorrhages, dark.

Myxoid cysts (colloid) differ from the former in having thicker walls, more copious vascular supply, and a differently arranged epithelium. The so-called glandular form (adenocystoma) is clothed internally with cylindrical epithelia in constant development, arranged in acini and resting on a vascular basement membrane. They may be multilocular or contain imperfect septa, or the proportion of the wall may be so great that the cavities appear as simple fissures and spaces in it, though clothed with epithelium. The contents of the cyst is thick and gelatinous, though in old tumors it may be serous; it contains pseudo-mucin, which forms a slimy solution in water and is not precipitated by acetic acid.

FIG. 31.



PAPILLARY CYSTOMA OF THE OVARY. (*Ziegler.*)

a, stroma; *b*, stratified epithelium; *c*, longitudinal sections of papillary outgrowths; *d*, transverse sections.

The *papillary form* of ovarian cysts is often observed in both organs at once. It has a layer of ciliated epithelium and is arranged in papillary projections on its inner surface; secondary cysts are less common, hemorrhage is more frequent, concentric masses of calcium carbonate (psammoma bodies) are more common both in the stroma and the epithelial collections, than in the glandular form; pseudo-mucin occurs but less copiously. The *glandular form* may arise from Pflüger's glands (remnants of the fetal mesothelium) or hyperplasia of the Graafian epithelium; they are less malignant than the papillary tumors. The latter arise from parovarian inclusions in the ovary or from the Graafian follicles. The genesis of neither form is accurately determined. Papillary tumors may form a metastasis to the peritoneum or become carcinomatous.

Dermoid cysts of the ovary are either loosely attached by a pedicle or are larger and supplant the ovarian tissue. They are common and affect one ovary. They may be teratomata, made up of all three embryonic layers and their products, or more strictly "dermoid," like similar tumors of other parts. They contain hair, fat, cholesterin, sweat glands and teeth in the simpler forms, and bone, cartilage, gland tissue, suggestions of organs of special sense (as the eye) and tissues resembling that of the nervous system in the more complex; for these tumors the name *fetus in fetu* is perhaps appropriate, though they have been explained as parthenogenetic developments from unfertilized ova.

Carcinoma of the ovary may develop in the walls of a glandular cystoma or start in the epithelium of the follicles; it rapidly invades the peritoneum in some cases.

The Fallopian Tubes.

Congenital or acquired *stenosis* of the tubes may be partial or complete; common causes of the latter form are inflammatory adhesions which compress one tube or keep it sharply bent, and tumors in the uterine wall. The result of closure of the part is usually the collection of muco-serous fluid in it (*hydrosalpinx, hydrops tubæ*). The distention often proceeds from the outer to the uterine end and dilates the part irregularly. The internal pressure drags the muscle fibers apart and causes atrophy of them and of the mucosa. The weight of the swollen tube may cause lateral and other displacements of the uterus. The fluid, which is clear and thin in most cases, may escape by way of the uterus.

Hyperemia occurs with peritonitis and metritis or is passive with impeded flow in the vena cava. Slight hemorrhages may occur from the tube during menstrual congestion, emptying into the peritoneal cavity or the uterus. The most copious tubal hemorrhage is associated with pregnancy and rupture in the part.

Inflammation.—*Salpingitis* may be catarrhal and either acute or chronic; it is usually associated with similar diseases of the uterus. Localized inflammatory adhesions often result between the tubes and the bladder, ovary or intestine.

Suppurative salpingitis is the result of gonorrheal or puerperal infection; in the former the infection may be mixed, the mucosa is hyperemic and thickened, the epithelia desquamate and the contents of the tube become purulent. If the pus is not emptied it may remain for a long time (pyosalpinx) and again become active or rupture as the result of later injury. In the puerperal form the inflammation may begin at either end, both may be closed by the edema and inflammatory exudate, and the fimbriæ inverted; the suppuration may involve the entire thickness of the wall and lead to rupture, either into the general peritoneal cavity, a hollow viscus, or an artificial space formed by adhesions. As in the other form, the pus may remain in the tube and thicken, mineral matter may deposit in it. Combinations of salpingitis and pelvic peritonitis and cellulitis are common and the abscess thus formed may be of large size. The pyogenic agent is often the streptococcus, but the pus may be sterile.

Tumors.—Benign forms are not of great importance or of large size; they may be fibroma, myoma, lipoma and papilloma, usually projecting into the tube but also presenting externally in some cases. Malignant tumors occur as the result of extension from the uterus and ovary.

Tuberculosis is not infrequent as a secondary lesion, with peritoneal and pulmonary affections, far less frequently with other genital tuberculosis. The disease begins at the fimbriated end of the tube and proceeds toward and into the uterus, miliary tubercles occurring in both serous and mucous coats. Chronic forms are accompanied by the usual infiltration with caseous softening. The tube is usually adherent to neighboring organs, swollen, thickened and distended, its mucous coat destroyed in places by caseous ulcers and the lumen filled with cheesy and purulent material. Primary tuberculosis of the tube does occur, often on a basis of gonorrheal inflammation. Both tubes are commonly involved in either form.

Mammary Glands.

Malformation.—With imperfect sexual development the breast may be absent or hypoplastic; the gland may be normal but lacking a nipple; supernumerary mammæ (polymastia) occur on the abdomen, back, thigh and elsewhere, in either sex, and the extra breasts may have or lack a nipple; they at times functionate.

A certain amount of congestion is very apt to occur with menstruation and the close sympathy between the organ and the genital system is shown also by premature development and function of the breast with premature sexual maturity, atrophy of the gland with cessation of sexual life, pain and swelling when the uterus is the seat of inflammation and other lesions. Other causes of hyperemia of the breast are injury and inflammation. Hemorrhage occurs after severe contusions and may be situated in the connective tissue or in the gland tissue; if the bleeding takes place into a lacteal duct it may dilate it and appear at the nipple. The extravasation may become surrounded by fibrous tissue (blood cyst). With tumors of the part bleeding is usual, both in the neoplasm and on the ulcerated surface.

Inflammation seldom occurs in the mammary gland except with puerperal infection, but the normal swelling of the gland which occurs at puberty in both sexes may pass into a mastitis, with swelling of the axillary nodes, and lesions of the ribs (caries) and the skin (erysipelas) may result in the same. Puerperal mastitis may be conveyed through the blood current (metatastic form) or enter the gland through the milk channels, and slight injuries to the nipple during suckling predispose to the latter form. The entire organ may be involved or only a section of it; the usual result is abscess formation and when the inflammation exceeds the limits of the organ paramastitis also occurs. The abscess, which may be single or multiple, may open into the milk channels, the tissues about the gland or externally (fistula formation). The abscess cavity slowly fills and heals by granulation from the sides. Rupture into the pleura with fatal empyema is very unusual.

Hyperplasia of all the elements of the gland may lead to enormous development of one or both breasts (*giganto-mastia*), without effect on the general health, but, if functionating, to corresponding production of (normal) milk. A hyperplasia limited to the fibrous tissue of the organ (diffuse fibroma) may preserve or reduce the size of the breast with extreme hardness and cysts of the milk channels. A circumscribed fibrosis occurs and may return after operative removal.

Myxoma may develop from the connective tissue of the organ as a diffuse tumor; the epithelium usually atrophies and disappears. If the neoplasm follows the milk ducts it is called intercanalicular myxoma.

Sarcoma of the breast may be diffuse or nodular. In the diffuse form the gland rapidly enlarges, usually but one, the process involves the skin and other tissues beyond, the gland becomes immovable and ulceration begins on the surface. On section parts of the tumor resemble fibrous tissue, other parts are myxomatous, others are soft and vascular; cysts from degenerative softening and from dilatation of the ducts occur, often of small size and containing clear or bloody fluid. If the cysts are an important feature of the tumor and chiefly from dilated natural cavities the name cysto-sarcoma is given to it. An unusual form of this tumor occurs when the sarcomatous process follows the milk ducts and distends them, the new tissue being covered by a layer of gland cells. The circumscribed sarcomata usually start in the ducts near the nipple and after a period of very slow growth become diffuse. Sarcoma starts in the adventitia of the ducts or the vessels or the interstitial fibrous tissue. Metastasis occurs through the blood-vessels and is not so pronounced a feature of the disease as in carcinoma.

Adenoma of the mamma is commonly combined with fibroma, lipoma, myxoma or sarcoma. It causes hyperplasia of the ducts and occurs chiefly as an encapsuled tumor, less often without being circumscribed. The epithelia of the neoplasm may be larger than normal but typically arranged in the acini, or with more rapid multiplication they fill the lumen and undergo degenerative changes.

Carcinoma of the breast is a frequent disease in women, between the years of 40 and 50, and apparently connected with the end of sexual life; the right breast is somewhat more frequently affected than the left. It occurs also in the male in about two per cent. of all cases. Injury is sometimes given as the preceding cause by the patient, but in many cases this is a coincidence, rendered important by the growth of a tumor later; in other cases a preceding inflammation seems to prepare the way for the neoplasm, or a benign tumor becomes carcinomatous (fibroma and adenoma). A special form known as Paget's disease begins as papillary dermatitis (eczema) of the nipple and its vicinity and carcinoma develops later in the same area; it has been claimed that the "eczema" is in itself a carcinomatosis of the epidermis.

All varieties of carcinoma and combinations with other forms, and degenerative changes in the tumor when formed, are encountered in the breast. They begin as hyperplasia of the epithelia in the acini of the gland (acinous form) or in the larger ducts (tubular form), and at first strongly resemble a simple adenomatous process. But the cells soon become very numerous, lying in several layers on the membrana propria, and in form they differ from typical epithelia in proportion as the disease advances; the limiting membrane is soon broken through and for a time its spindle cells may be recognized in the now atypical acini. The masses of cells are arranged in sinuous columns and smaller series through the gland, lying in the distorted acini and between them and invading the adjacent structures, as the pectoral muscles and afterward the pleura and lung. These masses of cells in a loose communicating mesh work are separated by a stroma much infiltrated with round cells; and in some forms the fibrous stroma increases steadily, contracts, and atrophies the epithelia, so that narrow spaces are left between its trabeculæ containing a few cells or their detritus. The usual degeneration observed is fatty, but myxomatous changes occur in either the connective tissue or the cell masses and calcareous deposit is common. Hemorrhage occurs in the softer forms, and, with ulceration, from the surface, but is not so much a feature of the disease as in sarcoma.

The chief varieties are:

1. Carcinoma simplex, in which the disproportion between stroma and cells is not marked, the tumor is not so soft as the medullary nor so hard as the scirrhus, and is less malignant than the former and more than the latter.
2. Medullary carcinoma, rich in cells, rapid in growth, the section yields much "cancer juice," hemorrhage may occur, ulcerates quickly and extensively; inflammation may coexist with abscess formation (mastitis carcinomatosa); malignant.
3. Scirrhous carcinoma, made up chiefly of fibrous tissue, slow of growth and relatively not very malignant; hard, little juice, nipple contracted to complete inversion; may ulcerate but ulcer also of chronic character.
4. Colloid carcinoma, by degeneration of either of the previous kinds; the change is often limited to but one portion of the umor; rare form.

5. Cystic carcinoma, from dilatation of glandular acini, the contents thin or jelly-like or fatty; unusual variety.

Metastasis is common and may be extensive with mammary carcinoma, the lymphatics of the axilla and clavicular region being successively involved and distant parts like the bones (vertebræ, pelvis), and adjacent organs, as the liver, pleura and lungs, being secondarily attacked. A local "regional" invasion is spoken of when secondary nodules form in the skin and tissues near by with intervals between. Microscopic secondary nodules in the skin are very frequent and explain recurrence in the scar after operative removal of the growth.

A form of epithelioma of the surface of the breast, with fairly rapid destruction of the surface and a narrow progressing edge of epithelial neoplasm, may occur on the breast as on other parts; it is often called rodent ulcer because of its gross appearance and slight resemblance to a neoplasm.

Cysts may be the result of occlusion of a milk duct and retention of the secretion; they may be small and multiple or fewer and larger; the contained fluid may be milky or serous, or thick and caseous.

Tuberculosis of the breast occurs with general disease of this nature and is a possible source of transmission to infants, for the bacilli pass out with the milk. It usually appears as a localized and chronic lesion and the breast commonly loses its function and atrophies.

Syphilis seldom attacks the breast but gummatous formation and its scars may be found.

APPENDIX TO CHAPTER VII.—DISORDERS CONNECTED WITH PREGNANCY.

Extra-uterine Pregnancy.—When the ovum is impregnated at the ovary, after the follicle has burst, a so-called *ovarian pregnancy* may occur, but is one of the rarest forms of the condition. *Tubo-ovarian pregnancy* is somewhat more common, the developing embryo being in part covered by the tube, in which the placenta usually forms.

Tubal pregnancy results from bending and partial occlusion of the canal which hinders the impregnated ovum from reaching the uterus; it is more common than the former. The chorionic villi grow into the mucosa of the part and an imperfect placenta forms from its vessels; the site of the fetus may be at either end of the tube, and with any variety it is common for a partial formation of decidua to occur in the uterine cavity. The embryo may die early or reach the third month; at that time the tension usually ruptures the tube, with bleeding into the peritoneum, which is worst when the placenta is torn, and danger of death from loss of blood or from peritonitis. When the embryo with its membranes leaves the tube it may form attachments to the peritoneum and be converted into a stony mass by the deposit of lime salts (lithopedion).

Abdominal pregnancy may be primary when the ovum settles on some part of the peritoneum and begins to develop without entering the tube; or secondary after exit from the ruptured tube.

Lesions of the Fetal Envelopes and Placenta.—Fleshy moles result from hemorrhage into the decidua, coagulation of the blood and degeneration of the tissues; adhesions between the fetal envelope and the placenta may form. The fetus dies and the mass is expelled or calcareous changes occur. Placental fragments which remain in the uterus may lead to formation of polypoid adenomatous growths; commonly after abortion. A destructive placental polyp is due to the hyperplasia of the placental elements and the invasion of the uterine wall by round, spindle and large epithelial cells.

Syncytioma malignum, or malignant deciduoma, doubtless takes origin in a manner similar to the foregoing. These tumors contain two varieties of cells, large epithelioid elements which are polymorphic and seem to multiply without mitosis, and smaller polyhedral cells which contain glycogen and multiply by mitosis; the former are regarded as coming from the syncytium and are frequently grouped in large masses and meshwork with coagula between them; the others are derived from the chorionic villi and lie in masses between the first variety. Transitions between the two are common. Metastasis occurs through the blood current, in the lungs frequently, the liver and the genital organs, and in this respect the tumor resembles a sarcoma; its epithelial elements suggest relationship with carcinoma. It is best to regard it as a mixed tumor like other forms which start from fetal inclusions.

Hydatid moles develop from edematous chorionic villi with hypertrophy and myxomatous changes, or from active hyperplasia of these structures with similar degenerations. They occur in connection with general anemic conditions (nephritis, chlorosis), entail the death and destruction of the fetus, and usually resemble a bunch of grapes, little cystic clusters hanging together by a few pedicles. Microscopically they consist of epithelial cells covering edematous or mucoid connective tissue, with few cells and some free nuclei; necrotic and calcareous changes occur in them.

The placenta may be hypoplastic or hyperplastic; it may be inserted over or near the cervix (*placenta previa*); its form may vary from the usual round cake, and small supplementary placentas occur. The attachment of the cord may be at the margin and if wide and fan-like is called a velamentous insertion; the cord may be partially double and knots and twists are fairly common.

Hemorrhage into the placenta occurs, and if large may cause death and expulsion of the fetus; hemorrhage from the placenta is a dangerous complication of placenta previa. So-called "infarcts" in it may be due to coagulation necrosis during the later weeks of pregnancy; they are yellowish-white areas made up of fibrin and round cells with red blood-cells.

Inflammation of the placenta may be diffuse fibrosis or localized nodules; the new tissue is often arranged about the vessels. The entire placenta may be indurated and either loosened in its attachment to the uterus or more firmly fixed. Placentitis may be due to extension from endometritis.

Tuberculosis and syphilis may affect the placenta, the usual gross and microscopic lesions developing as in other tissues. A syphilitic taint is assumed in many cases of placentitis. Fatty and calcareous degeneration of the placenta may occur.

Many of these conditions lead to the death of the fetus at various periods of pregnancy. It may then be expelled, or remain in the uterus for a long time and undergo putrefactive changes and as the result of these the mother may become septic.

CHAPTER VIII.

DISEASES OF THE LOCOMOTOR SYSTEM.

Osseous Tissue.

Hyperemia is normal in the bones of the new-born and during their growth; in completely formed bone the marrow is yellow, except in ribs, sternum and pelvis, where a red color persists till late adult life. Pathologically, hyperemia is active with inflammation, passive when the venous return is impeded; if chronic the marrow cells increase and the fat is absorbed. The gross appearances of the bone are redness of periosteum with swelling and edema, dark or bluish-red color in the marrow, with hemorrhages in some cases.

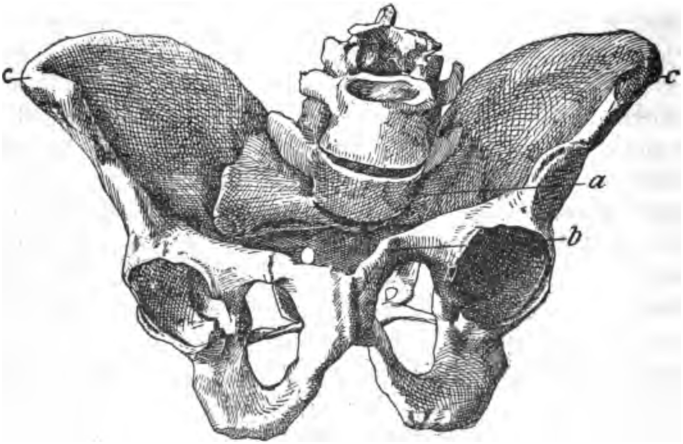
Thrombosis occurs with fracture and from neighboring inflammation, but produces but little alteration because of free venous anastomosis. *Embolism*, except of the main nutrient artery and when septic, may also cause no extensive lesion. *Hemorrhage* follows injuries to periosteum and bone tissue, but the blood is usually absorbed rapidly unless infection occurs, as in compound fractures. When a large *hematoma* forms under the periosteum the bone may undergo superficial necrosis from disturbance of its nutrition. One case of such hematoma is found in the new-born, the blood being extravasated under the scalp and periosteum, usually confined to a single bone, as the parietal, from injuries received during protracted and instrumental deliveries; a corresponding hemorrhage between dura and inner surface of the same bone may occur. Usually the blood is absorbed without delay. Caries and new growths are other causes of hemorrhage in bones, especially vascular sarcomata,

Rhachitis ; Rickets.—This is a nutritive defect in the skeleton connected with insufficient supply of mineral substance to the bones, most always observed in the early years of life (first to third) and associated with anemia, leucocytosis, swelling of the spleen, and intestinal catarrh. While the essential cause of the condition is not definitely known, it may be that the chief factor in its causation is excessive formation of lactic acid in the alimentary canal, imperfect absorption of lime salts, and deficient alkalization of the blood, together with imperfect oxidation in the tissues. The excretion of mineral matter by way of the kidney may be excessive, but usually it is below normal. All parts of the skeleton, and both the periosteal and the medullary aspects of the bones, show imperfectly ossified tissue, and the usual arrangement in concentric layers in the long bones is imperfect or absent. In the epiphyses the deposit of lime salts is irregularly distributed or lacking. In some cases there is large formation of osteoid tissue under the periosteum, especially at tendinous and aponeurotic insertions. The marrow is generally red and rich in cells, resembling the fetal condition; the vessels at the epiphyseal ends may be actively congested. The enlargement of the ends of long bones is due to the hyperplasia of cartilage cells, increase in size in the medullary spaces, and development of osteoid tissue, together with the effects of gravity and pressure. The common deformities which result are bendings of long bones (infractions), swollen ends in ribs and limbs, large cranium with prominent forehead and open fontanelles, spinal curvatures (*kyphosis* if backward, *lordosis* if forward, *scoliosis* if lateral; these may be combined), and prominence of the sternum (pigeon breast). In the pelvis the deformity is important in relation to child-birth; the commonest change, from muscular traction and superposed weight, is shortening of the superior conjugate diameter and lengthening of the transverse. When the sacrum is specially prominent the inlet has a trefoil outline.

A prenatal rhachitis occurs in which the bones are uniformly thickened and shortened, with large ends, bendings and adipose hyperplasia under the integument, and sometimes cretinism from premature union of the cranial sutures.

Osteomalacia is a disease of adult life, commonest in women in connection with parturition, in which there is rapid and general resorption of the inorganic salts from the bone. A mild degree of decalcification appears to be frequent in puerperal women, and the disease has been regarded as a trophoneurosis associated with pathological excess of this process. It occurs in certain regions especially (along the Rhine). It may be limited so certain portions of the body (pelvis) or begin thus and involve other bones in succeeding pregnancies. The prognosis is almost invariably bad.

FIG. 32.



OSTEOMALACIA OF THE PELVIS. (*Ziegler.*)

a, last lumbar vertebra, displaced forward; *b,* horizontal ramus of pubes, bent backward; *c,* iliac crests.

Spongy bones suffer most but the lesion, with fractures and deformities, is not confined to them. In the pelvis the pubic arch and the sacral promontory are pushed forward, the iliac crests outward and the sides of the pelvis inward, resulting in a beaked and triangular pelvis. Owing to spinal tenderness the patient lies habitually on one side, with consequent unilateral excess of the pelvic deformity. Fractures are common and multiple, occurring spontaneously or with slight injuries, and as many as thirty in one patient have been recorded. As the process begins in developed bones its structure is retained for a time even when soft enough to cut, but in fractured parts and bent bones osteoid tissue is common, not in regular arrangement. The fractures show a tendency to repair even during the activity of the process of resorption.

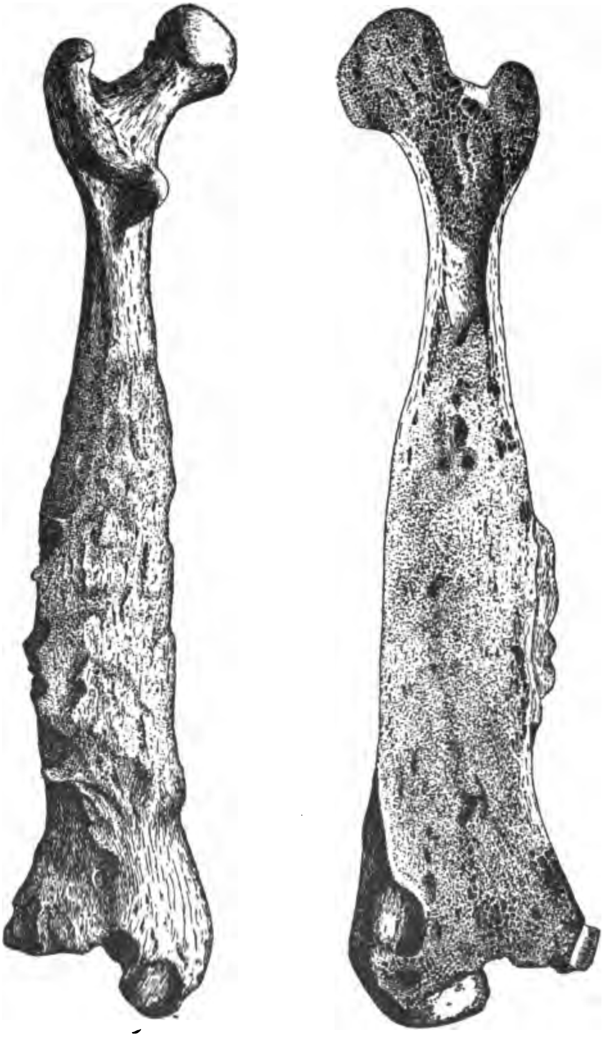
In the gross the bones are soft, may be freely bent and twisted, cut easily with a gritty feel if still containing salts; layers of osteoid material under the periosteum and in the medullary spaces are found, the marrow is congested, fatty or gelatinous; calcified and soft parts may be sharply distinguished from each other or gradually fade from one into the other. Microscopically the Haversian canals are widened, new fissures form, the decalcified tissue is fibrillar with perhaps a trace of lamellar arrangement, and the bone corpuscles are atrophic. The marrow may be hyperemic, rich in cells, or in a condition of mucous degeneration. Osteoid tissue may contain large bone corpuscles.

General cachexia sooner or later appears and the patient dies of pneumonia or other intercurrent disease.

Inflammation.—*Periostitis* may be simple, suppurative or productive (ossifying). Simple or fibrous inflammation results from contusions and other non-infected wounds and produces general thickening of the membrane, with firmer adherence to the bone; inclusions of foreign matter may result in such inflammation long after the injury is received. The vicinity of chronic ulceration (leg) and joint affections commonly results in such fibrosis. The periosteum appears hyperemic and swollen and infiltrated with round cells.

Ossifying periostitis is a chronic process which results in the production of exostoses, osteophytes and bony thickenings. It may invade connective tissue adjacent (parosteitis). The usual

FIG. 33. FIG. 34.



CHRONIC OSTEITIS AND PRODUCTIVE PERIOSTITIS. (*Coplin.*)

Surface of bone under periosteum nodular and thickened, marrow cavity nearly obliterated.

condition with which this form is associated is chronic inflammation of joints, syphilitic and other disease of bones, but it may appear with general constitutional diseases (rickets) and physiological states (as bony plates in the dura with pregnancy). The toxins of chronic tuberculosis seem at times sufficient irritation to cause it.

Purulent periostitis may result from infection through the blood or directly from bone disease; pyogenic organisms of all varieties are found. A circumscribed form with abscess under the membrane may lead to exfoliation of a thin sheet of necrosed bone with perforation externally and extrusion of the sequestrum. At times the condition is so general and destructive that the name *periostitis maligna* is given to it. The process may lead to suppuration in the medullary canal or result from it, and in either case the danger of septicemia and pyemia is marked.

Osteitis and Osteomyelitis.—These two conditions can hardly exist separately. The cause is usually infection and the course is either acute or chronic, the former producing abscesses and the latter a fungous condition from excessive granulation tissue, abscess and necrosis. Acute traumatic inflammation follows fracture with infection, amputation and other injuries, and causes hyperemia and swelling of the marrow, diffuse infiltration with pus, and abscesses where this is most evident. Necrosis and sequestrum formation, perforation of the pus into joints or outwardly, and pyemic infection may be associated with this form. Acute infectious (spontaneous) osteomyelitis occurs chiefly in the long bones, hemorrhages may occur, the epiphyses may separate or even an entire bone undergo necrosis. In less extensive cases single abscesses may form in one bone. Pyemia is the common end of the worst cases.

Chronic inflammation of the bones is usually associated with preceding acute disease of the part, wounds, syphilis and tuberculosis; specific forms show the usual caseous changes. A rarefying form is described (*osteoporosis*) and a condensing form (*osteosclerosis*); the former is accompanied by absorption of the walls of Howship's lacunæ by osteoclasts, which begin as small cells and end as giant cells, sometimes with processes, which cause chemical destruction of the gelatinous elements of the bone and absorption of the lamellæ. A reparative or reactive osteosclerosis is often associated with the foregoing and may cause deposit of new bone under the periosteum (eburnation) and also fill the medullary canal with it.

Necrosis.—Embolism of arteries, separation of the periosteum, inflammation of various portions of the bone, may lead to such nutritive disturbance that a portion of it dies; this process is called necrosis and the dead piece of bone, the *sequestrum*, is partly softened and absorbed, the remainder lying as an irregular eroded body in a cavity lined with granulations and partly filled with pus. The pus makes its way through the thickened periosteum and discharges on the surface through one or more fistulæ or sinuses, bits of dead bone coming away also. The granulating periosteum begins to form new bone about the sequestrum, which lies as a foreign body under it, but complete repair can follow only when the sequestrum is cast off. In necrosis of the central parts of the bone this is usually difficult, except when it occurs in a stump after amputation, a ring of bone with lateral projections into the narrow cavity being often formed and easily removed. With chronic phosphorus-poisoning necrosis of the bones of the face, most commonly the lower jaw, begins in the periosteal aspect of the bone and is accompanied by production of new osseous tissue. Suppuration is common and may lead to pyemia. After operative removal the patient may recover with much distortion of the parts. The entrance of phosphorus vapors and pyogenic organisms is facilitated by caries of the teeth.

Tuberculosis of Bones; Caries.—Miliary and other forms of tuberculosis occur in the bones, before birth, in childhood, and in adult life with lung lesions, but less frequently. The disease is hematogenous, the bacilli brought through the blood-current, and secondary to tubercular lesions of the lymph nodes (scrofula) and the organs of the thorax and abdomen; in the child, portions of bone in most active development are involved and previous injury is often recorded. The disease often begins in spongy tissue at the ends of long bones and opens into the joints or in the small bones of the hands and feet. In the gross there are small grayish or yellowish foci, singly or in groups, surrounded by a margin of active hyperemia. These increase in size, become confluent, and undergo caseous softening of their centers, the portion of tissue involved may separate from the still healthy bone by a line of demarcation, and within it occurs the molecular death of the tissue known as caries, in distinction to the large sequestra of necrosis. The focus of softened bone, mixed with pus, forms a tuberculous abscess, its walls covered with a pyogenic membrane. The combination of rarefying osteitis, granulation-tissue formation to replace the lost bone, and destructive suppuration of this, progresses in the line of least resistance and opens into joint cavities or externally usually by multiple sinuses. The pus focus in rare cases becomes encapsuled, thickened and healed. Should perforation of the pus follow the lines of muscles and fascia (as in caries of vertebræ) the sinuses lead for long distances through the tissues and permit the accumulation of large amounts of pus; to these the name cold abscess is applied, and in psoas abscess, one of the commonest, the entire integument above the knee may be elevated from the muscles and converted into a sac filled with pus. It is especially in the long-continued suppurations connected with chronic osteitis that amyloid degenerations of the viscera occur.

Syphilis.—Congenital syphilis occupies especially the epiphyseal lines of the long bones, appearing as a pale yellow jagged line about 2 mm. broad; this increases in width as the cartilage adjoining it becomes swollen and gelatinous; later the fatty degeneration of the cells between the line and the bone permits the easy separation of the epiphysis. The spleen is commonly enlarged in this disease.

Acquired syphilis causes periosteal growths, gummatous infiltration with softening and necrosis, leading to large defects in the bones (as of the skull), and condensing osteitis.

Leprosy causes severe ulceration of the soft parts and necrosis of the underlying bones (*lepra mutilans*) which ends in loss of large portions of the extremities. Other bone lesions in this disease are osteomyelitis and granulomata with giant cells and lepra bacilli in the marrow.

Atrophy.—The process of absorption and replacement of bone is constantly present during life; in marasmus, cachexia, senility and lack of use the replacement may reach so low a proportion that the bones atrophy, generally or in special regions. The localized atrophy occurs from pressure, as with aneurysm and tumors; the general form occasionally occurs in young subjects, often in more than one of a family, without apparent explanation. General atrophy is also called *fragilitas ossium.*

In certain injuries the bones of a part, as a limb, become useless, and in early life they may remain behind in development (hypoplasia); as a congenital condition this hypoplasia produces dwarfs (*microsomia*).

Hypertrophy as a general condition of the bones, seems, like general atrophy, to be connected with trophic nervous centers; the most marked instance of this is acromegaly, a pathological giant growth developing most often about puberty, leading to excessive size of the limb bones, extremities, and face. With this condition syringomyelia and lesions of the pituitary body have occurred but a causal relation has not been established between nervous lesions and the condition.

Local hypertrophies occur as exostoses, osteophytes, and exercise bones; some of them are due to irritation of the periosteum, and some to hyperplasia laterally from epiphyseal cartilages. If chiefly composed of cartilage the tumor is called *exostosis cartilaginea*, if of bone, *osteoma.*

Tumors.—*Fibroma* and *myxoma* may affect the periosteum; chondroma and osteoma are the commonest benign tumors of bone.

Sarcoma occurs as a primary tumor in most of its forms, peripheral or central, including spindle-celled, round-celled, pigmented, angiosarcoma and giant-celled. The growth causes resorption of the bone, breaks through the periosteum and reaches a large size; where the periosteum is preserved a new deposit of bone goes on from its under side so that the tumor is enclosed in a thin shell. It may grow slowly for years, without affecting joints or making metastases or causing much pain. General sarcomatosis of many bones may occur but this is usually secondary to a distant neoplasm. Microscopically the cells are distinguished from marrow cells by their uniform size, their finely granular protoplasm, the appearance of spindle cells, and rapid invasion of the tissues. A special variety of sarcoma of the periosteum of cranial and facial bones is called *chloroma*, from its green or yellow color. Primary carcinoma of the bones probably never occurs; as a secondary tumor all varieties have been observed. Epithelioma of the skin may directly invade subjacent bone, with progressive absorption. Embolic cases of multiple development have been called carcinomatous osteomalacia.

Cysts occur in bones almost entirely from softening of other neoplasms, as myxoma.

In leukemia and pseudo-leukemia the marrow may present numerous small lymphoid tumors, called *lymphoma* or *myeloma*.

Joints.

Injuries and Displacements.—Fractures may lead into joints. Pieces of articular cartilage may be broken off and remain as foreign bodies in the joint cavity (floating cartilages, as in the knee). Complete solution of the relations of bones at joints is called dislocation; incomplete forms are called subluxations. The capsule of the joint and one or more of the ligaments are torn in such injuries, as also the tendons, muscles and other structures about the joint. When the relation of the bones is restored healing in the capsule and ligaments rapidly follows by productive inflammation. If unreduced, the end of the bone suffers partial absorption, ossifying periostitis makes a new false joint and fairly good movement may be possible; the torn muscles and ligaments atrophy. In other cases the bones are united more or less firmly by fibrous, cartilaginous or bony tissue, with practically no motion preserved and no attempt to form a joint; the condition is known as ankylosis. It occurs also as one of the results of inflammation in joints.

Hyperemia of a joint may be the only appearance at autopsy corresponding to acute rheumatic affections in the early stages and after contusions. The edges of the cartilages and the synovial membrane present swollen vessels or even hyperplastic increase (*synovitis pannosa*) and the fluid of the part may be increased.

Hemorrhage into joint cavities (*hemarthros*) is usually the result of injury, but it occurs also in hemorrhagic small-pox and other constitutional conditions; the blood may be absorbed or leave pigment granules in the tissues.

Edema (*hydrarthros*) occurs with inflammation, almost never with hydremic conditions.

Fibrinous arthritis may be "dry" or accompanied by extravasation of serum. It exists as one of the lesions of articular rheumatism, or with inflammation in the vicinity of the joint, or after traumatism. The fibrin lies in the fluid as white or yellowish flocculi or as a film clinging to the synovial surfaces. With the addition of pus the forms of arthritis known as seropurulent and fibrino-purulent occur; it is commonly the case that all three elements appear together, as with infectious diseases. The synovial membrane is thickened and swollen and covered with soft layers of purulent fibrin; all the tissues of the joint are infiltrated with pus-cells. If the joint contains chiefly pus the name arthropyosis or abscess of the joint is employed. The common result is the destruction of the cartilages, and caries of the articular ends of the bones; the ligaments and tissues about the joint are also involved (*panarthritis*). After the pus escapes, healing with ankylosis may occur, or chronic inflammation persists with its danger to life from pyemia and amyloid degeneration. Such joint abscesses at times complicate typhoid fever, diphtheria and dysentery and are common in pyemia.

Chronic arthritis includes several forms of inflammation of the joints with secondary atrophic changes and others in which degeneration is more evident than inflammation. The etiology may be the same for the two; preceding inflammation, injury, anomalies of metabolism, senility and certain central nervous conditions are the chief factors. Chronic arthritis appears both with and without exudation.

Chronic serous arthritis (*hydrarthros*) follows serous and serofibrinous acute lesions or begins as a chronic condition, as in the knee. The synovial membrane is swollen and opaque, its edges proliferate, and the joint contains a varying amount of serous fluid. The usual course is a transition to one of the other forms of chronic arthritis.

Chronic suppurative arthritis is the result of infection through the blood or from abscess in the neighborhood; it may follow acute suppuration and is common with tuberculosis of the joints. The synovial membrane is thickened and covered with purulent fibrin, the cartilages undergo fatty degeneration, their basement membrane becomes fibrillary; necrosis follows and exposes the bone, which then becomes inflamed. In favorable cases the joint becomes rigid as the process is healed.

The non-exudative forms of chronic arthritis include arthritis deformans, arthritis sicca, arthritis adhesiva, a fungous form with tuberculosis and uratic or gouty arthritis.

Arthritis deformans, chronic gout or rheumatoid arthritis, includes chronic degenerative with productive lesions, and results in such alterations as to merit the name deformans. Its course covers years and usually begins in late adult life. The first changes are in the cartilages, the basement membrane becomes fibrillary and the cells proliferate actively; afterwards fatty degeneration destroys the cartilages, especially at the sides of the articular surfaces. The bone thus laid bare becomes hard and polished, but already, under the remaining cartilage, the bone has undergone marked absorption. The ligaments, at first thickened, become contracted and fibrous, and, together with the production of new bone about the edges of the joint, help to produce complete ankylosis. The absorption permits luxations, and the ankylosis often forms afterward. The hip, knee and finger joints in this way may present every variety of dislocation and every angle of ankylosis, rendering the patient entirely helpless.

Arthritis sicca differs from the preceding but little; it is common in the aged, attacks the hip, shoulders and elbow in the order of frequency, and is not connected with such destruction of bone and production of new osseous tissue. The arthropathies seen with tabes dorsalis and syringomyelia resemble arthritis deformans of more than commonly slow progress and may involve only the lower extremity.

Adhesive arthritis follows acute and other inflammations and leads to the union of joint surfaces by the production of organized fibrous tissue starting in remnants of cartilage, especially where the amount of exudate has been small. Many joints may be affected at once. From the large amount of vascular new formed fibrous tissue the process is sometimes called arthritis pannosa.

Arthritis uratica, gout, podagra, follows from the deposition of urates in the fibrous connective tissues about joints, in the cartilages and the bones, and commonly affects the small joints of the hands and feet, especially of the big toes. Similar deposits occur in other places. Beside the joint lesion there are commonly visceral and vascular changes associated, fatty heart and liver, chronic interstitial nephritis, gravel and calculi in the urine, tophi in the external ears, and arteriosclerosis. The course of the disease is very chronic, marked by periods of a few days of acute and painful inflammation of the joints (the paroxysm) and months of apparent health. While the etiology is not definitely understood, the disease attacks by preference those who live well, consume wines habitually and take but little exercise. The soluble quadrurates circulating in the blood become changed into the insoluble biurates and are deposited about the joints, perhaps with some relation to the habitually low temperature and the exposure to injury of these parts.

Tuberculosis of the joints follows rupture of a tuberculous bone focus into the cavity, or it begins as a primary fungous synovitis (synovitis granulosa). While bacilli are present in all acute cases, it is sometimes difficult to find them, except by inoculation of animals from the joint contents. The joint affected is swollen, edematous and altered by luxations. On section the tubercular masses may be found as gray or yellow points. The joints may be filled with profuse granulations, or they may have suffered purulent softening and the bone is the seat of caries; cold abscesses developed in this way may remain till the joint is practically disorganized or burrow and empty on the surface. Where the joint supports weight, as in tubercular diseases of the vertebræ (spondylitis) various deformities result, as kyphosis. The disease frequently develops in children, with or without injury to the joints, and the larger articulations are specially liable to invasion, as the hip, knee and elbow. Favorable results with ankylosis are obtained in some cases, but the position of the limb is seldom normal. "White swelling" was the name formerly given to this disease in the knee.

Syphilis in the congenital form thickens the capsules and ligaments of various joints, or a gumma near by may soften and involve a joint, or syphilitic osteochondritis may become a purulent lesion and involve the joint and the tissues about it. In adults gummata may form in joints and by their softening and scars produce an arthritis deformans syphilitica.

Tumors.—From the synovial fringes small lipomata and fibromata develop and on separation they may be free in the joint; another and more numerous newly formed body in joints is the so-called rice-like bodies, which occur also in bursæ, fifty or more at a time. They probably arise from hyaline portions of the synovial membrane. Other neoplasms occur in connection with bone lesions.

Muscles.

Anemia is part of a general condition or results from local pressure and loss of circulation; the muscle is pale and dry at first, later it shrinks and becomes friable.

Hyperemia is physiological with exercise, it occurs with inflammation; stasis is unusual. All these conditions are difficult to appreciate on the dead subject owing to the changes of the death agony and rigor mortis.

Hemorrhages occur from tears and contusions, and after degeneration of the tissue, as with waxy muscles in typhoid. Thrombosis and embolism are not important here.

Inflammation, myositis, follows injury and infection; the organisms may come from the vicinity or by the blood (pyemia).

Acute myositis with serous exudate, inflammatory muscular edema, is accompanied by an edematous affection of the connective tissue between the muscle bundles with cellular infiltration to a moderate degree, while the muscle cells remain nearly passive or degenerate, with vacuole formation and hyaline changes. It follows injury, external or caused by trichinæ and anthrax. The fluid may be absorbed, or purulent and hemorrhagic features be added.

Acute primary polymyositis is spoken of when many muscles at once are attacked, without apparent cause. The muscles swell and became extremely painful and tender, and the skin over them may be edematous. The position of the muscles involved is important; if those of respiration and deglutition are affected death may speedily result, with pneumonia. The muscle is pale or streaked with yellow, and under the microscope shows irregular cell infiltration between the fibers and the bundles. The striation of the muscle is lost, fatty and granular changes occur and fissures in the muscle fibers. The apparent resemblance to toxin effects is noticeable but no bacterium has been discovered.

Secondary acute myositis may occur with infectious disease (variola, typhoid and diphtheria).

Hemorrhagic myositis may be the result of extension of diphtheritic inflammation from the margins of a tracheotomy wound, or result from extension of phlegmonous inflammation of other parts. Scurvy and serpent venom may also cause it.

Acute suppurative myositis usually occurs as abscesses in the interstitial connective tissue. Their origin is traumatic, embolic and pyemic, or cryptogenic. A phlegmonous form results from severe skin lesions and extension to the muscles below. The results differ with the infection; in mild cases the perimysium offers an obstacle, in others the suppuration invades the muscle and abscesses form.

Chronic myositis may be a slow purulent lesion without apparent reaction (cold abscess) with chronic tuberculosis, actinomycosis and glanders; or it may be a slow productive lesion, with increase in the fibrous elements and atrophy of the contractile (*myositis fibrosa*), as after acute myositis, after neuritis and other nervous lesions and in the vicinity of chronic bone and joint diseases; or, thirdly, it may be of the ossifying type, localized at tendinous insertions, from prolonged activity of the muscles (exercise bones, etc.), or a progressive myositis ossificans which sometimes occurs in the young after severe falls, chilling of the surface and sudden efforts, involving the muscles of the back and neck and resulting in wry neck and other deformities from permanent shortening.

Tuberculosis of muscles occurs as cold abscess in connection with joint and other lesions; other forms are unusual; even with general miliary tuberculosis the muscles are commonly not affected.

Syphilis causes a fibrous myositis, with contractions, and usually involves one or but few at a time (masseter, biceps); or it occurs as gummatous infiltration, sometimes of a single muscle, as the sternomastoid of one side.

Glanders invades the muscles as multiple small foci or nodules with a tendency to suppurate and break down. It may extend from the mucous membranes, as of the mouth, or be generally disseminated.

Atrophy may be simple, myopathic or neuropathic, according as it depends on defective nutrition, disease of the muscles, or central nerve lesions.

Simple atrophy occurs in old age or when muscles are not used for long periods, as in fractured limbs in splints.

Primary myopathic atrophy may develop in childhood and there appears to be a strong hereditary history with many cases. Several muscles at a time may be involved, usually in the lower extremities, as the calf. The apparent size of the part may be larger than normal owing to the fatty replacement (lipomatosis), but the muscle cells are atrophied, with here and there a large fiber. No demonstrable changes in the nervous system occur in this form, which is one of the chief reasons for separating it from the next.

Muscular atrophy of neurotic origin may result from the most diverse lesions in the cord; syringomyelia, lateral sclerosis, descending degeneration after cerebral lesions and multiple neuritis, are some of the conditions in which the muscles atrophy in the territory of distribution of affected nerves. So-called progressive muscular atrophy as a clinical entity begins often in the small muscles of the hand, proceeds to involve those of the forearm, upper arm, shoulder, diaphragm and back, and may not at all affect those of the lower limbs. Atrophy and degeneration of the muscle fibers, fibrosis of the interstitial tissue, and fatty degeneration in the nerves of the part (motor) are the usual conditions found. The anterior nerve roots and the anterior horns of the cord may also present evidences of degeneration with atrophy of the ganglion cells. This is most marked in the cervical enlargement, corresponding to the seat of the lesion.

Hyaline or *waxy degeneration* of the muscles occurs in the course of infectious diseases, especially typhoid fever, and while any muscle may be involved, even the heart, the rectus abdominis is most often the seat of the process. In the gross the muscle looks dry and glistening, with stripes of granular or hyaline degeneration having a waxy look. Microscopically the fibers are first swollen and then homogeneous and dull; transverse fractures occur, as also in the gross; hemorrhage may take place into the tissue of the muscle. Fibers not waxy present granular changes and the interstitial tissue is hyperplastic. Of the many other diseases besides typhoid in which this change occurs, may be mentioned burns and freezing, delirium tremens, progressive muscular atrophy, neoplasms invading muscles, trichinosis, small-pox and relapsing fever.

Fatty degeneration occurs with myositis and any cause which deprives the muscles of nutrition. Two special forms are worth mentioning, that occasioned by phosphorus, which seems particularly to affect the trunk muscles, and a similar condition in the new-born, chiefly limited to the lower extremities.

In obese persons a form of fatty infiltration occurs.

The most important parasite in muscles is the trichina already described in Pt. I., p. 195.

Tendons.

Inflammation (thecitis, tendo-synovitis) follows injury or extends from suppurating foci along the course of the tendon. Its course may be acute or chronic and the exudate may be chiefly serous, fibrinous or purulent; the results are much the same as in the case of arthritis with exudate.

Tubercular disease of the tendons usually results from similar lesions of bones and joints in the vicinity. The sheath of the tendon is usually much thickened and in the secretion rice-like bodies are often found in numbers.

Ganglion is the name given to a collection of fluid in the sheath of a tendon, or in a hernial protrusion of it, and the fluid is serous or more gelatinous.

Bursitis affects principally those bursæ which are situated in front of bony prominences subject to constant pressure, as in miner's elbow, coachman's bursa, housemaid's knee. As is evident from the popular names, the condition is a lesion of occupation. In the old days of the hand loom, "weaver's bottom" was a common complaint, from inflammation of a bursa over the tuber ischii. The inner surface of the thickened bursa is usually smooth, lined with flat cells, or roughened, and the fluid is either serous or purulent. Fine vegetations occur in the chronic form and rice bodies (*corpora oryzæ*); these are regarded as tubercular by some writers.

Tuberculosis and tumors involve the bursæ and tendons from the accident of vicinity.

CHAPTER IX.

SPECIAL PATHOLOGY OF THE SKIN.

DISEASE processes are the same here as in other organs but their effects, owing to local conditions, are much more varied. The skin is not only exposed to extremes of heat and cold to which other viscera are not subject but it is the habitat of many parasites from whose invasion the internal organs are free. Moreover its structure is complex and the same morbid condition in different situations may produce pathological and clinical pictures of wide variance. Even in the same location, a toxic agent may cause several alterations which have little in common. It is necessary to recall one point in physiology, that the sweat glands secrete a fluid fat, in addition to water holding various salts in solution, while the sebaceous apparatus produces only a solid fat for the lubrication of the hair.

As they recur constantly, it is well to begin with a perfect understanding of the cytological terms employed here. In addition to the ordinary elements found in exudation, the polynuclear leucocyte, eosinophile and lymphocyte there is at least one other of importance, the plasma cell. This last is a derivative of the lymphocyte probably and shows a round, eccentric nucleus whose chromatin stains in clumps, not diffusely, and basophile protoplasm (methylene blue) more dense at the periphery. The cell is ovoid or polyhedral from pressure. Cells derived from the fixed connective tissue corpuscles are of several sorts; the fibroblast with oval, vesicular nucleus, branched or spindle-shaped body whose protoplasm is granular and generally slightly basophilic; the pseudo-plasma cell (sometimes called connective-tissue phagocyte) which differs from the plasma cell in being larger and in having a homogeneous protoplasm with affinity for acid dyes; the epithelioid cell which is oval, acidophile, with a large vesicular nucleus and the mast cell whose origin is doubtful. The last occurs in bizarre forms but it has a distinctive characteristic in that its granular protoplasm exercises a selective action on the red in Unna's polychrome methylene blue. Giant cells, also fibroblastic in origin, are of two types, the myeloid whose nuclei are scattered through the cell body and the Langhans form, in which nuclei occur in a peripheral horseshoe or ring.

For convenience, the classification used by the American Dermatological Association will be followed here, omitting those disorders whose rarity makes their consideration suitable only to works on dermatology, and others, like the neuroses, which leave no demonstrable histological traces.

Anomalies of Secretion.—Anomalies of secretion comprise those evidenced by changes in composition and in amount, increase or decrease. Cysts are not strictly part of this category but may as well be considered here since the secretion forms the tumor.

Bromidrosis is due to contamination of the eliminated sweat by the bacillus fetidus, the offensive odor being due to the by-products of its activity. *Chromidrosis*, blue, red or green sweating, owes its appearance to various chromogenic organisms. *Uridrosis* is the result of the elimination through the skin of the urinary salts. Suppression is not a necessary precedent. *Hematidrosis* occurs in hemophilia and in psychical disturbances where stigmata appear, blood-corpuscles or hemoglobin being present. *Hyperidrosis* is often congenital and may display a striking unilaterality, occasionally in the distribution of a single nerve. Many observations point to a purely nervous origin for it. *Anidrosis* is never total and occurs in the course of systemic disease. *Seborrhea* was formerly thought to be an anomaly of sebum secretion but is now recognized generally as a part of seborrheic dermatitis. So far as is known there is no qualitative change in the product of the sebaceous glands.

Sudamina, *hidrocystoma* and *miliaria* are retention cysts occurring along the tract of the sweat ducts. Sudamen and miliaria are superficial, hidrocystoma rather deep in the corium. Sudamina occur in the course of fevers, are located chiefly in the epidermis whose layers are separated by the fluid and are accompanied by little inflammation. Hidrocystoma has a thick covering composed of the whole epidermis with part of the papillary layer. The duct is enormously dilated in its middle third as a rule and the cavity may be lined by flattened cells. Miliaria is the ordinary "prickly heat," and differs from sudamina in being an inflammatory lesion. All three occur in conditions of excessive moisture and high temperature, hidrocystoma almost exclusively on the faces of washerwomen and cooks. The sweat duct has no lining of its own in the epidermis, consequently the moisture causes the cells to swell, the lumen is finally blocked and a cyst results, superficial or deep as the obstruction occurs in the horny layer or among the prickle cells. In long periods of tropic heat miliaria may become pustular, pyococci finding a suitable medium in the warm, sodden skin. They may be introduced from outside or living in the organ, like the staphylococcus epidermidis albus, merely awake to activity. Fatty, sudoriparous cysts occur outside of these conditions, in males at the time of puberty. They are probably due to a congenital defect of patency in the sweat apparatus. The water excreted is doubtless reabsorbed, leaving a semifluid fat. The cysts are subject to infection. Ducts have been traced into all these cystic formations.

Milia and sebaceous cysts differ as do sudamina and hidrocystoma, in their location in the skin. The former never attain great size and are superficial; the latter are large and deeper. Both are caused by obstruction, congenital or acquired (scar), in the sebaceous opening. The epithelial cells of the lining are flattened or disappear and the solid, fatty mass is enclosed in a thick fibrous capsule. The pressure of the cyst causes atrophy of surrounding structures and thinning of the epidermis. The contents may calcify after a lapse of years.

Inflammations.

Urticaria.—The characteristic lesion of urticaria is the wheal, due to rapid outpouring of serum from the superficial vessels. In susceptible individuals the vasomotor apparatus is in a state of unstable equilibrium and responds at once to slight stimuli, external, internal and reflex. On the first application of the stimulus the vessels dilate and a large amount of serum is exuded, relatively to the area involved. If the tissues are loose, the lesion remains red ; otherwise, the fluid in the interstices causes a compression of the blood channels and the center becomes white. In children, rarely in adults, the exudation may be sufficient to raise the epidermis and form a bulla. The efflorescence requires only a few minutes for its evolution and its subsidence may be quite as rapid, so that there is no opportunity for alteration in the epidermis or proliferation of fibroblasts. Serum is not the only blood element exuded. In dermographism, or factitious urticaria, there is an extravasation composed entirely of lymphocytes which is present in a slight degree in the ordinary form. Urticaria is hemorrhagic at times from diapedesis of red corpuscles. The mechanism of giant urticaria or angioneurotic edema is exactly the same as in the smaller wheal, the area involved reaching great size.

Erythema.—This term is applied to a number of conditions, but here its meaning is confined to Hebra's erythema exudativum multiforme, a name which describes the condition closely. Multiform erythema is ultimately allied to urticaria and purpura rheumatica. Its basis seems in most cases to be a condition identical with that known as acute rheumatism. This erythema is a symptom of a systemic intoxication which may produce also gastro-intestinal crises and acute nephritis. Pyococci have been cultivated from the skin and blood in many cases. Drugs often cause an erythema.

The histology of this lesion is simple. There is dilatation of the vessels of the corium and an extravasation about them, composed chiefly of polynuclear leucocytes with an edema much less marked than in urticaria as a rule. There is little alteration in the epidermis but in persistent cases there may be some overgrowth in it. In the papular form, leucocytic invasion is the striking characteristic; in bullous erythema, edema is sufficiently great to raise the horny layer; in herpes iris, the bulla subsides in the center, leaving a ring peripherally, containing fluid; in erythema nodosum, there seems to be a total vessel paresis, the lesion is blue from stasis, boggy from edema and on subsiding leaves discolorations like a bruise. In addition to organisms often present, there is an eosinophilia of the serum in the bullæ which is very rarely found in the blood. In purpura or peliosis rheumatica, in addition to the usual picture present in erythema, there is extensive cutaneous hemorrhage, red corpuscles occurring intact or partly destroyed. Hemorrhage occurs in the course of erythema not infrequently as the result of administration of salicylates, a condition which should not be mistaken for purpura in which hemorrhage is the chief symptom.

Scaling Dermatoses.

In inflammations of longer standing than the two just considered, when the process is of a subacute or chronic character from the beginning, changes occur in the epidermis which are characteristic of all cutaneous catarrhs, moist or dry. All of them are directly attributable to the presence of an increased amount of serum, an edema, of the epithelial layer. The first change is a widening of the intercellulur spaces, intercellular edema or spongy metamorphosis, as Unna calls it. The second is an intracellular edema, due to imbibition of the fluid by the cells which favors a retention of their juicy, granular character normal in the lower layers up to the surface, the clear and horny layers having disappeared. The edema prevents a proper transformation of the cell-protoplasm through keratohyalin into the keratin of the horny layer and the cells, instead of desquamating as in normal conditions, stick together and a scale is formed. This process is called parakeratosis, is present in every scaling disorder and is not to be confounded with hyperkeratosis in which the horny layer is greatly thickened but the cell transformation is complete. The third phenomenon is overgrowth of the rete. Its interpapillary projections are widened or lengthened or both and the whole layer is increased in depth. Mitoses are to be found in the germinal or palisade cells. The condition is called acanthosis. All are secondary to inflammation of the corium.

Eczema may with entire justice be defined as a catarrhal dermatitis, exudation being evident in its dryest, clinical phase. The stimuli responsible for an outbreak of eczema are, even now, little known. They are certainly nervous reflexes in some cases, autointoxications (gout) in others. Microörganisms complicate the process always when it has been in existence for a short period, but it has never been proved that the primary vesicle of the disease contains bacteria, much less that they have anything to do with the causation of eczema. "Parasitic eczemas," so called, are best relegated to the class of seborrheic dermatitis or else regarded as contaminations.

In spite of its varied clinical forms, the process is always the same. There is first congestion, then edema and exudation in the corium, particularly the papillary layer, the exuded cells at first sheathing the vessels, but spreading later in a uniform mass throughout. The cells are chiefly lymphocytes and polynuclears. Proliferation of fibroblasts is present in acute outbreaks and is a marked feature in old patches. Eczema exerts its marked effects on the epidermis where intercellular edema, parakeratosis and acanthosis are present in different degrees, their combinations giving rise to the diverse clinical pictures. In acute vesicular eczema, this fluid collects in droplets beneath the horny layer, into which pus-cocci find their way and cause tiny abscesses. In eczema madidans the fluid is poured out in such quantity that the horny layer is washed away completely, leaving a sodden rete from which drops exude to run down or dry on the surface. Eczematous crusts are formed of fibrin, cell detritus and numberless organisms. The last stage of every eczema is desquamative. The scaling is a result of the parakeratosis and the scales themselves are composed of partially cornified cells, in which nuclear remains are often to be seen. Acanthosis is shown in the thickening and lengthening of the interpapillary projections and in increase in depth of the whole epidermis.

Seborrheic dermatitis (Unna's *eczema seborrhoicum*) differs histologically not at all from true eczema but its etiology is different. A great deal of painful labor has been expended on this question of causation. It is hard to frame a clear-cut statement which will not mislead. I believe that no one will deny the presence in the earliest lesion of the morococcus. This is a minute, round organism which stains readily with methylene blue, may be cultivated on ordinary media and occurs in colonies, not unlike the appearance of the mulberry from which its name is taken. It has been reinoculated with success, producing characteristic lesions, so Unna says. It is found in the horny layer, the vesicular fluid and lymph spaces. Accompanying it often is the flask bacillus of Malassez which is regarded as a modification of the morococcus, as both may be of the staphylococcus. Many investigators deny any etiological rôle to the morococcus in spite of its constant presence. Certainly, the disease is both hetero- and auto-inoculable, which eczema is not.

Psoriasis.—This disease approaches closely to scaling eczema and seborrheic dermatitis. In fact, there is no dividing line, in either a clinical or a pathological sense. Cell infiltration and fibroblast proliferation are less marked than in eczema of the same degree of acuteness. There is not a great deal of edema present. The histological condition is a dry catarrh, among whose effects parakeratosis is most prominent. The scales are thick and may show only partly keratotic cells throughout or the central portion may be edematous and the outer completely cornified. There is no stratum lucidum and the rete is thinned except for an increase in length of its interpapillary projections. The latter may pursue a tortuous course and may anastomose with neighboring rete pegs. The papillæ lengthened correspondingly with the epidermic prolongations covered by a thinned rete, reach close to the surface, scale, and their tips are often exposed when the scales are torn off. Nothing is known of the etiology of psoriasis.

Dermatitis exfoliativa occurs either as a sequel to other inveterate scaling conditions or in single or recurrent outbreaks as part of a systemic intoxication. The exciting agents include drugs and bacterial toxins, particularly those of pyogenic organisms and the tubercle bacillus. Leucocytosis is very slight as a rule, congestion is marked and edema evident, at least in the early stages. The epidermis is never raised in vesicle formation. The rete is edematous as in psoriasis, the granular and clear layers have disappeared and parakeratosis is less noticeable than in the conditions just discussed. Acanthosis appears, but to no great extent. In the *pityriasis rubra* of Hebra which succeeds repeated attacks of dermatitis exfoliativa, or persistent psoriasis, or arises spontaneously without antecedent scaling, nearly the same conditions are found. The disease invariably ends in death, usually through intercurrent tuberculosis; in its terminal stages, the whole of the epidermis and corium are thinned by atrophy of their cells and connective tissue. Congestion and fine desquamation remain to the end.

Pityriasis rosea is a superficial inflammation of very slight grade. There is congestion of the vessels of the cutis with a slight and irregular leucocytosis; the epidermis is thickened in its horny layer, the intermediate strata between it and the rete having disappeared. No organisms have been found.

Pityriasis Rubra Pilaris of Devergie is synonymous with the lichen ruber acuminatus of the Vienna school. Its first sign is a keratosis, an increase in the horny layer about the openings of the lanugo hair-follicles on the backs of the hands and the extensor surfaces generally. The cornification spreads only a short distance down the follicle. In the course of time, inflammatory phenomena appear in the neighborhood of the plugs, the vessels are dilated, a few leucocytes infiltrate and edema brings the usual changes in the epidermis. The disease may finally lead into pityriasis rubra of Hebra, in which case the whole surface becomes red and scaling and the perifollicular keratosis may disappear.

Lichen Planus.—The efforts to connect this disease with pityriasis rubra pilaris have failed. Its pathology is its own. In the earliest papule there is an infiltration of cells about the dilated vessels of the papillary layer only. The reticular layer is not affected. The cell infiltration is dense and its character is in dispute. The majority of observers are agreed that it is almost purely lymphocytic, while others assert that the cells are of connective-tissue origin and that the disease is a granuloma. The papillæ and epidermic projections are increased in length and the latter are also widened at the expense of the former. The horny layer is thickened, the rete cells are increased in number and flattened laterally by pressure of the infiltration beneath. In persistent cases hyperkeratosis may become so marked that the papule resembles a wart and is then called lichen corneus. In other instances, in addition to overgrowth of the epidermis, a process of organization goes on in the exudate, new vessels and new connective tissue are formed and the condition is termed lichen hypertrophicus. In involution, the infiltration disappears by degeneration and absorption and deep pigmentation as in syphilis occurs. Masses of granular pigment appear in the dilated vessels and are picked up by the lining endothelium. The granules are then passed on to wandering branched connective tissue cells resembling fibroblasts (chromatophores) and by them deposited finally in the cells of the germinal layer or left lying free in the spaces of the cutis.

Prurigo occurs in the tiny papules whose genesis is probably the same as for urticaria; in fact, chronic urticaria is in children likely to become prurigo in the course of time. The papule shows on section a general inflammation of the corium, the cells being chiefly lymphocytic and most abundant about the vessels. Edema is present in sufficient amount to cause a vesicle formation. The serum soon dries however and leaves a thickened epidermis capped by a blood crust, the result of injury. A peculiar feature of prurigo is cell infiltration of the arrectores pilorum and in older lesions a marked hypertrophy of their bellies. Fibroblast proliferation results finally in increase of the collagenous fibers of the corium.

Erysipelas is due to the presence in the cutis of the streptococcus of Fehleisen which is morphologically and culturally identical with the streptococcus pyogenes. It gains entrance through a solution of continuity and may be found in zoögleic masses in the lymph vessels. In the corium the process is an edematous and necrotic one; when it penetrates the subcutaneous tissue, it takes a frankly purulent character and becomes a cellulitis. The cutis presents a peculiar appearance. Its vessels, lymph and blood, are enormously dilated, their endothelium fairly intact and their contents showing a strikingly small number of leucocytes. They do not appear even in the neighborhood of the masses of cocci. The toxins not only destroy them but seem to exercise a negative cytotactic influence upon them as well. Outside the vessels, there is great edema, the tissue elements are widely separated and undergoing necrosis, the cells and the nuclei taking only acid dyes. Leucocytosis is as little apparent here as within the vessels. The epidermis is edematous and partly necrotic, its horny layer stripped off or raised in a large bulla. Tissue edema may cause partial collapse of dilated vessels.

Bullous Diseases.

Next to nothing is known of the etiology of this class of the skin's inflammations. A neurotic origin is imputed to them by almost all observers, in the sense that the word is used in urticaria, meaning a vasomotor instability of the cutaneous vessels. No changes have been demonstrated in the peripheral nerves or in the central system, with the exception of Kaposi's cases of pemphigus vulgaris in which sclerosis, which may have been entirely independent, was found in the posterior columns of the cord. These remarks do not apply to zoster in which the origin is now clearly understood.

Pemphigus Vulgaris.—There is very little congestion and leucocytic invasion of the corium. Serum is poured out of the vessels in a relatively great quantity for the time required in the production of a bulla. Unlike the vesicular conditions, where bleb formation is adventitious, the cover of the lesion is practically the whole epidermis. Remains of the rete pegs are often seen projecting downward into the fluid. In a recent bulla, the fluid shows fibrin filaments with a few leucocytes among which eosinophiles have by far the largest proportion. An eosinophilia is also found in the blood of a number of the patients. Polynuclear leucocytes appear with a coccic invasion which is always a possibility, though no part of the disease. In *pemphigus foliaceus*, the bullæ show the same appearance except that they are flaccid and soon rupture, the broken epidermis with dried serum and cells forming a ragged rim about the site of the bulla. In *pemphigus vegetans*, condylomatoid outgrowths appear on the bases of the blebs, as they do in other conditions. Their origin is probably to be found in local conditions of heat, moisture and bacterial activity.

Pemphigus acutus and *pemphigus neonatorum* together with the bullous eruptions which occur in the neighborhood of foci of bacterial activity might well be called, as they sometimes have been, septic pemphigus. They have no etiological relationship with true pemphigus, but are due to infections with microörganisms. The acute variety owes its origin to a coccus called by the name of its discoverer, Demme, which may be a modification of one of the ordinary pus cocci. Both it and the pemphigus of infants (a streptococcic invasion) appear in epidemics and may terminate in a general systemic infection. The histology of the bulla is the same in all forms.

Dermatitis herpetiformis is a multiform disease, occurring not only in bullæ but in erythematous, papular and pustular forms as well. It has been for many years, and is still in certain quarters, confounded with pemphigus without good grounds. Even more stoutly than the latter affection it is held to be of nervous origin, in spite of failure to demonstrate appreciable nerve lesion.

As a rule congestion is more marked in the cutis than in pemphigus, the leucocytosis of the papillary layer is more marked and the eosinophiles appear in greater abundance. The polynuclear leucocytes, lymphocytes and eosinophiles are chiefly found in the form of sheaths about the vessels. Intermingled with them are proliferated fibroblasts and mast cells. In the formation of a bulla the epidermis is lifted from the papillæ in its entirety and the rete prolongations are seen projecting into the fluid below. The contents consist of fibrin threads with a few entangled white cells, chiefly eosinophiles, the proportion of the latter reaching at times as high a figure as 60 per cent. There is almost invariably an eosinophilia of the blood. Leredde has claimed that the coincident increase in the eosin cells is characteristic of dermatitis herpetiformis. Scars are sometimes left, when the corium is completely denuded, and pigmentation as in lichen planus is always a sequel of the process. In pustular herpetiform dermatitis, the process is much more severe. The whole papillary and the upper reticular layers are packed with cells and there is a relative as well as an actual increase in polynuclear cells. Fibroblasts and mast cells occur in abundance. Cocci are found and in the purulent condition following their invasion, there may be partial necrosis of papillæ and rete. *Herpes gestationis* is a dermatitis herpetiformis occurring in the course of a pregnancy.

Pompholyx is a neurotic bullous affection of the palms and soles. In the author's patients, outbreaks have invariably followed shock and nervous depression. The disease has not, as was formerly thought, any relation to the sweat apparatus and in consequence is not related to sudamen or miliaria.

The process is inflammatory. There is the same mild leucocytic invasion about the distended papillary vessels as in pemphigus and dermatitis herpetiformis, great serous exudation with the formation of a bulla which occurs within the prickle-celled layer, not between it and the papillæ. Owing to the thick horny layer of the palms and soles, the fluid does not readily escape and the rete cells are flattened by the pressure. The fluid contains leucocytes (not a great proportion of eosinophiles) and epithelial detritus. Pustulation is a common accident.

Herpes facialis and *progenitalis* are neurotic inflammations which should not be confounded with herpes zoster. Their vesicles appear over an inflamed papillary layer, in the substance of the rete, the covering being formed of the whole horny layer. Owing to the sites, they become infected readily and, in such a case, necrosis is followed by a shallow ulceration. Very little scar tissue results from the healing process.

Zoster is the one affection in this category whose neurotic basis is thoroughly established. For years, opinion was divided between this theory and a vasomotor hypothesis. According to Head's investigations which have set all reasonable doubts at rest, the disease is caused by changes in the Gasserian or spinal ganglia; these may be degenerations but are more commonly interstitial hemorrhages. The active agent of their production is unknown, but it is probably an infection, since one attack usually protects for the individual's life. There may be acute neuritis of varying grade in the peripheral nerves themselves; in old people, the outbreak is often followed by an interstitial neuritis whose changes are of course permanent in the nerve and at times in the skin itself.

The vesicle of zoster appears in the substance of the rete with a sufficiently thick layer of epithelium below to account for the fact that few scars are formed. The vesicular fluid contains fibrin, leucocytes, a few eosinophiles, epithelial cells and in addition a number of minute, round bodies, free or enclosed in epithelial cells. They are homogeneous and have an affinity for acid dyes. Their resemblance to the so-called vaccine bodies is very striking and like them and similar bodies in carcinoma and molluscum contagiosum, they have been classified as protozoa. There is no fact to support such a theory. It is probable that they are merely the product of cell degeneration, hyalin in all likelihood. The upper part of the corium shows distended vessels, invested by leucocytes with a few lymphocytes. In healing, the vesicle dries into a scab, falls off and the horny layer is regenerated. If it becomes infected, ulcerations and scarring may result. Pigmentation is a fairly frequent sequel.

Pustular Conditions.

Sycosis may be of two varieties, bacterial and trichophytic. Only the former will be dealt with here. Sycosis is chiefly coccogenic; Mibelli's bacillogenic disease is excessively rare. The lesion is a perifolliculitis but there is an arbitrary regional division in the use of these terms. Sycosis occurs only in the beard and vibrissæ, folliculitis and perifolliculitis on the scalp and the hairy parts of the body. Favoring productive factors are heat and moisture. The infection is from the outside along the shaft of the hair. In a fully developed folliculitis, the hair is loosened from its sheath, partly disintegrated by the invading pus and covered by a mass of cocci, usually white staphylococci. The blood-vessels in the neighborhood of the follicle are distended and packed with red corpuscles. The connective tissue fibers are separated by edema and numbers of leucocytes, closely packed near the hair bag, the infiltration fading away until it becomes only a vascular sheathing at the periphery. A pus cavity is formed at the center which contains the remains of the follicle. The purulent detritus usually finds a vent at the surface, destroying the epidermis but at times it finds an easier passage to the subcutaneous tissue and collects in abscesses of considerable size. Partial destruction of the follicle entails no loss of hair but when the hair-papilla is lost, no new growth ever appears. Complete baldness results from folliculitis decalvans, a scalp disease, although pus formation is not so marked as in sycosis.

Furuncle.—A boil is a staphylococcic infection through the hair-follicle, like sycosis, but the result is very different, probably due largely to feeble resisting powers in the tissues. The process extends laterally and into the hypoderm almost at once. Large abscesses are not formed usually; the cocci find their way from the follicle along the lymph channels and edema and leucocytosis follow their course. Collagenous connective tissue is destroyed and the necrotic mass with leucocytes and bacteria forms the central "core" or slough which appears at the point of entrance and ramifies throughout the inflamed area. After its spontaneous separation or removal, healing by process of granulation takes place. *Carbuncle* is identical in its histopathology with furuncle but of greater extent and presents numerous sloughing areas.

Ecthyma is rather a consecutive lesion of the skin than a clinical entity since it occurs in the course of many diseases, notably those attended by pruritus. Through a solution of continuity in the epidermis, staphylococci gain entrance to the cutis, weakened in resisting power; the point of infection is oftenest produced by scratching and is independent of the hair-follicles. The beginning of the condition is seen in a pus collection in the papillary body, serum and leucocytes percolating into the epidermis. The abscess may be quite large and may raise the horny layer in the form of a pustule. After a time, the pus and epithelium dry into a thick crust which rests on the cutis, the surrounding skin intensely inflamed. The loss of tissue is always superficial and yet deep enough to heal only with scar formation.

Impetigo Contagiosa has been divided by Sabouraud into two varieties, those of Bockhardt and of Tilbury Fox, the first perifollicular and due to the streptococcus, the other not so situated and caused by the staphylococcus. The distinction is too fine for use. Location is probably altogether accidental and the staphylococcus albus and aureus the chief offenders. Unna says they have peculiar cultural characters in this condition and doubtless they have, for the infection is always mild. The streptococcus may be causative in the beginning of certain lesions and disappear before other pus-cocci later. In any case we are dealing with a superficial pustulation in the epidermis. The horny layer is lifted at the outset by a fluid collection which at this time contains few leucocytes. Later it becomes frankly purulent and dries with the covering epidermis in a crust which is stuck on, not set in the skin as in ecthyma. The base of the pustules is formed by a larger part of the rete and on subsidence of inflammation the stratum corneum is reformed without scar or pigmentation.

Acne is an inflammation about the sebaceous glands. In its site of predilection, the central portion of the face, the lanugo hairs are appendages of these structures, reversing the condition in sycosis in which the follicle is primarily involved. The basis of the acne pustule is the *comedo* which is not formed of inspissated sebum as was long held but of concentric layers of horny material. In other words, at the time of puberty when circulatory changes occur about all the skin appendages, one of the effects is a hyperkeratosis of the sebaceous opening. The outlet blocked, sebum collects behind the comedo and the gland is distended. A certain amount of the fatty matter penetrates the horny plug together with multitudes of bacteria. Sabouraud states that his "microbacillus of seborrhea" is the chief factor in comedo formation but the investigations of others have not supported his contention. The pus organisms are white and golden staphylococci and the streptococcus. Remaining quiescent for a time, they finally cause congestion in the periglandular plexus, followed by a leucocytic invasion. When the latter is of light grade, inflammation may resolve without destruction of the gland, but a small abscess is usually formed which causes destruction of the appendage and finally points about the opening. This is called acne vulgaris. In the worst cases, especially when the skin is thickened as the result of previous inflammation, instead of breaking through the skin, the pus burrows beneath it, and several lesions coalesce into subcutaneous abscesses. The process is then called acne indurata.

Acne varioliformis occurs about the border of the scalp anteriorly, is a necrotic perifolliculitis, a central slough separating with the hair as in furuncle and leaving a depressed scar much like the pits of variola.

Dermatitis papillaris capillitii (acne keloid) occurs on the neck at the edge of the scalp, in persons, negroes especially, in whom a keloidal tendency is marked. The beginning lesion is a pustular perifolliculitis, which is followed by a formation of new connective tissue in thick bands parallel to the surface. Coincidentally, there is a dilatation of the vascular channels into large angiomatoid spaces which may result from obstruction of outflow by the new keloidal growth.

Rosacea.—This process has nothing to do with acne; the name acne rosacea is a misnomer. It is divided into three stages, hyperemia, pustulation and hypertrophy. The first evidence of the disease is a dilatation and lengthening, causing tortuosity of the superficial vessels. There is slight edema of the tissues and evidence of subacute inflammation in the shape of an emigration, chiefly of lymphocytes. Later, pyococci gain entrance through follicles or sebaceous glands and small abscesses are formed, infiltration becoming leucocytic. The abscesses may be independent of all the appendages. In the third stage, the long-standing edema stimulates the activity of all the cells, fibroblasts and epithelium. The sebaceous structures are greatly hypertrophied and surrounded by dilated vessels and new collagenous fibers. Pustules dot the surface here and there and the whole cutis is infiltrated by polynuclear leucocytes and lymphocytes, with fibroblasts showing occasional mitosis and a few mast cells.

Hydradenitis Suppurativa.—It is likely that hydradenitis is the same process as the one called acne varioliformis and various others to which no less than eleven appellations have been given. The author prefers to call it necrotic granuloma as indicating its nature and committing the description to no particular appendage. The precedent conditions are a persistent acroasphyxia and often, though not always, systemic diseases, among which tuberculosis is easily first. The inflammatory process begins deep in the reticular layer and subcutaneous tissue, about the plexuses of the sweat coil (hydradenitis), follicle (acne) or independent of them both. It is almost impossible to suppose an external infection for such a condition. The nodules are at first composed chiefly of lymphocytes and plasma cells with a few leucocytes; later, epithelioid and giant cells appear and the nodule undergoes a central necrosis which, if not coagulation necrosis, is nearly allied to it. Neighboring foci coalesce until, by extension, the epidermis is involved and exfoliates; the necrotic mass is separated. As there is loss of substance, there is always minute scarring as a sequel.

Atrophies.

Alopecia Areata which has no relationship with any other of the many forms of baldness is really an inflammation of light grade, but as its chief symptom, pathological as well as clinical, is loss of hair, it is best considered here. Sabouraud claims that it is due to the same microbacillus which he regards as the causative agent of seborrhea and acne, as well, but, as in acne, he has found no supporters in alopecia areata. Microörganisms are present in numbers early in the disease, but, according to Robinson and Walker, they are the staphylococci epidermidis albi of Welch, slightly modified in its cultural characters. An areate alopecia has been produced by it in rabbits. There is very little inflammation present about the follicles. Interference with hair growth seems to be caused by toxin acting on the hair-papilla. The hair is at first thinned, then expands again so as to produce the characteristic exclamation point appearance (!). When the dot is formed, the hair is lost, leaving the follicle empty. After a time, new growth begins in the form of a white, lanugo, tapering hair, the lack of color due as always to loss of medullary pigment, not presence of air in the shaft. Later, hairs appear of the normal size and color. In total alopecia, after the formation of a few lanugos, the papillæ may atrophy, the hair bag disappear and baldness is permanent.

Scleroderma.—There are two forms of the disease, a localized (morphea) and a generalized one which belongs to the domain of neuropathology. Localized scleroderma begins in the form of a vascular dilatation accompanied as in keloid by a perivascular proliferation of fibroblasts. These fibroblasts produce new collagenous fibers which appear in thick bands and in turn cause an obliteration by pressure of the blood-vessels. Finally, the blood-channels appear as hyalin threads and failure of nutrition produces an atrophy of all the cutaneous structures, the terminal stage of the disease.

Hypertrophies.

Increase in the thickness of the horny layer or interference with its proper formation produce various lesions which may be classed as *keratomas*. Among them are cutaneous horns, corns, callosities, seed warts, keratosis pilaris, a relative of ichthyosis (*vide infra*) and other conditions difficult to classify. The dividing line between tumors, hypertrophies and inflammations is a very narrow one.

Condyloma acuminatum results from an overgrowth of the entire epidermis, evidenced by increased thickness of horny layer and rete, and mitotic figures in the germinal cells. As a result, the epithelium is thrown into projections, into the concavities of which the papillary vessels and connective tissues follow. The vessels are enlarged but contrary to what might be expected, there is little or no evidence of fibroblastic proliferation. This papillary formation is rarely simple, offshoots appear on the parent stem, giving finally a cauliflower appearance. Condylomata acuminata appear in favoring conditions of warmth, moisture and uncleanliness (bacterial invasion?) notably about the genitals.

Molluscum contagiosum, if not actually a contagious affection, is certainly anto-inoculable. In spite of many efforts, no organism has ever been recovered from its tumors. The rete is the portion chiefly affected. The horny layer is lost in a fully developed lesion and an opening leads down to the center of the mass which partly projects above the surface and partly into the corium. Its lower border bounded by connective tissue is often lobulated in the fashion of sebaceous glands. The germinal cells are reduced in height and the neighboring rete cells have largely lost their prickles. The characteristic feature of molluscum contagiosum is a degenerative process which appears in the mucous layer. Small areas of degeneration appear in the cells, which take eosin readily and are possibly composed of hyalin material. They increase in size and coalesce until the whole cell body is occupied. These are the "molluscum bodies," so-called, which like all similar degenerations have been held to be protozoa. They lie free in the central cavity and may be extruded by pressure as a milky fluid. (Pt. I., p. 243.)

Ichthyosis.—There can hardly be any question that ichthyosis is a congenital deformity of the horny layer. Unna, in one of his vagaries, classes it among infectious inflammations. The fact that it is rarely seen before the end of the first year is readily explained by the tendency of infants' skins to exudation under slight irritation which the process itself supplies. Bowen's theory is the most tenable yet presented—that it is the result of the persistence of the fetal epitrichial layer of balloon cells. Histologically, the chief change is an increase in thickness of the horny layer with a marked thinning of the rete. Although the secretions are noticeably lessened, there is no perceptible change in the number, size or appearance of the sweat and sebaceous glands. The papillæ are elongated and taper to a point. Ichthyotic skin is susceptible to all forms of irritation; the type of the resulting inflammation is eczematous. Consequently, there is usually present in the corium a greater or less degree of subacute, catarrhal inflammation, evidenced by the presence of lymphocytes, leucocytes and fibroblasts. This mild grade of ichthyosis is called xeroderma; ichthyosis hystrix shows thick plates and spines of horny material not unlike the ectoskeleton of the saurians. The keratotic layer is apt, like the tip of the comedo, to become blackened in time, either from dirt ground in or a change in the character of the keratin.

Pigment Hypertrophies appear spontaneously, independently of inflammation or new growth, in the shape of freckles (lentigo) or moth-spots (chloasma). The change is the same in both, an increase of a non-ferruginous pigment contained in the germinal layer and in chromatophores wandering through the corium.

Elephantiasis occurs in two forms, the Arabic and the Greek as they have long been called. Both result from obstruction of the lymphatic circulation, but the blocking is due to different causes. In elephantiasis Arabum, it is caused by the presence in the lymph radicles of the ova of the filaria sanguinis; in elephantiasis Græcorum by a variety of scar-producing conditions, syphilis, tuberculosis, surgical operations, etc. When the obstruction is set up, the lymph vessels dilate and a portion of the fluid is exuded into the surrounding tissue where it stimulates the fibroblasts to proliferation and formation of new connective tissue. In the process of contracture, the new fibers press upon lymphatics and veins and new ones are occluded. The vicious circle thus set up causes ultimately an enormous increase in the size of limbs, scrotum or vulva as the case may be. The epidermis is thickened, verrucous in places, and deeply folded. Here and there the epithelial layer becomes thinned from the pressure of a lymphangiectasis below and finally ulcerates, permitting the escape of a milky fluid. (Pt. I., p. 197.)

Hemorrhages.

Purpura is symptomatic of many diseases and injuries of the skin. It is evidenced histologically by the pressure of red corpuscles or hemoglobin granules in the lymphatics and between the fibers of the corium. Inflammation may or may not be present. The term is not applied to bullous or vesicular lesions which have become hemorrhagic. Purpura occurs after direct violence and from bites of insects such as the body louse, in the course of almost all inflammations, exceptionally, such as syphilis and variola, in scurvy (purpura hemorrhagica) and with or without erythema as purpura rheumatica. The last is generally the result of a pyococcic infection, is accompanied by inflammatory changes, and in severe cases there may be localized necrosis followed by ulceration. Extravasation of blood is due to one of two causes in the skin as elsewhere, the presence of a hemolytic ferment or weakness of the capillary walls. In *purpura senilis*, where hemorrhage occurs spontaneously, Unna has demonstrated changes in the endothelial lining with thrombosis and rupture of the capillaries.

Granulomas.[1]

Syphilis.—Chancre, the initial lesion of syphilis, results beyond question from the entrance through a break in skin or mucous membrane of a microörganism which has hitherto eluded all research. The claims of Lustgarten, Van Nieson and others of the discovery of a bacillus have never been substantiated. The first change is a great congestion, followed by an exudation composed almost entirely of plasma cells (see introduction to this chapter) and lymphocytes. They first appear closely packed in sheaths about the small vessels, so far as can be judged from the advancing border. The lesion is always necessarily far advanced when excised for examination. The pressure which the plasma cells exert upon each other causes them to assume a polyhedral shape, as also the few epithelioid cells dotted throughout the exudation. The connective tissue, collagen and elastin, largely disappears but threads may be traced traversing the cell mass. At the periphery, the rete prolongations are increased in size but over the center they have disappeared as a result of pressure, the rete is thinned and the horny layer often exfoliated. The vessels of the corium show changes of various grades from a mere swelling of endothelial cells to a panarteritis with proliferation of the lining sufficient to occlude the lumen. Remains of the capillaries can be seen forming the center of almost every focus of emigrated cells. It is possible that outside pressure has also something to do with capillary obliteration. The immediate neighborhood of the vessel, deprived of its nutriment, undergoes necrosis. Under a lower power, the field can be seen dotted with the pale foci of degeneration, if only a basic stain is used (polychrome methylene blue). In addition to the cells described, a few mast cells may be found at the edge of this infiltration and in exceptional cases, here and there, a Langhans giant cell with protoplasmic filaments extending into the surrounding tissue. Chancre is not in itself an ulcerative process; when ulceration occurs it is from a superadded infection from pus-cocci or the bacillus of chancroid. Necrosis begins beneath the epithelium, spreading to it and the tissue below. Numbers of polynuclear leucocytes appear near it. After a time, the necrotic material separates and an ulcer with sharp edges is left. The extreme hardness of chancre is due solely to the density of its infiltration. When the process of repair begins, the cellular elements undergo fatty degeneration and are absorbed, leaving numbers of fibroblasts which produce an amount of collagenous material totally incommensurate with the needs of the occasion. This constitutes the usually permanent scar of chancre.

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[1] See also Pt. I., pp. 234–245.

Papular Syphiloderm.—As the papule forms the basis of all early lesions of syphilis, one description of it will suffice. Ulceration and pustulation do not alter its character sufficiently to prevent its recognition. It is well to remark that in the absence of tubercle bacilli, differentiation is impossible between it and disseminate tuberculosis. The papule begins like chancre in the form of an exudation of plasma cells and lymphocytes about the dilated, congested vessels of the cutis and hypoderm. With the exudation, there is a proliferation of adventitial fixed cells which appear chiefly in the epithelioid form. A small number of mast cells are to be found at the periphery. As the lesion grows older giant cells are formed here and there. The vessels show endothelial proliferation which obliterates them at times and causes focal necroses. The epidermis is flattened and thinned over the densest part of the infiltration, its rete pegs increased in size at the periphery. Resolution is the same as in chancre with a resultant pigmentation whose mechanism does not differ from that described under lichen planus.

Chancroid has at last been established as a definite disease by the cultivation and inoculation of its bacillus. The organism is short (streptobacillus of Unna), occurs in chains in the tissues and irregularly in the necrotic detritus. It was discovered by Ducrey and cultivated on serum-agar first in 1896 by Lenglet, whose work has been confirmed. It is recovered with great difficulty from the pus either of the sore or the bubo. There are many ulcerations about the genitals due to pus infection which are not chancroids. The bacillus gains entrance through broken epidermis to the papillary layer where it forms a small abscess, the course through the epidermis marked by leucocytes surrounding bacillary chains. They are never seen in the protoplasm of phagocytes. Outside the abscess is a zone of plasma cells not so densely packed as in chancre from which lines of emigrated cells radiate, following the course of the congested vessels into the hypoderm. At the edge of the plasmoma a few fibroblasts and mast cells are found. The epithelium is edematous and infiltrated with pus. Necrosis begins early and superficially, destroying the epidermis and corium beneath so that the edges overhang. In a fully formed chancroid, the surface is covered with débris containing many leucocytes and bacteria bounded by successive zones of necrosis with bacilli and fragmented nuclei, the plasmoma much reduced in thickness and peripherally congested. Leucocytes migrate through the whole diseased area. The floor is uneven, due to the fact that tissue death occurs first in the neighborhood of the chains radiating from the ulcer into the edge of the plasmoma. Bacterial activity is finally overcome by the leucocytes, probably, since their number constantly increases, and, after sloughing or absorption of necrosed tissue, healing takes place by scar formation.

Tuberculosis.—Tuberculosis verrucosa cutis is the commonest form in this country and is due to a direct inoculation, generally of the hands, with Koch's bacillus which is usually found with comparative ease. The whole cutis and hypoderm is the seat of a diffuse tuberculosis. The chief cellular elements are plasma cells, lymphocytes, epithelioid and giant cells, the last derived from fibroblasts whose protoplasm has not kept pace with nuclear division and usually containing the few bacilli to be found. There is no regular arrangement of the cells into typical tubercles. Threads of connective tissue run irregularly through the mass. Necrosis is uncommon. At the base of the elongated projections are found numerous miliary abscesses. They may be caused by pyococcic contamination but the organisms are diffcult to find. There is a great degree of acanthosis, the rete pegs are elongated, sometimes club-shaped or digitate at the extremity, the lower border perhaps lost in an abscess. The horny layer is enormously thickened by hyperkeratosis and raised into the verrucous formation which gives the disease its name.

Lupus Vulgaris is a tuberculosis of lower grade and slower growth. Bacilli are demonstrated with difficulty by scraping the tissue and smearing it on a slide. The lesion of lupus is the "apple-jelly" nodule which is always to be found. It consists of small cellular areas in the cutis and hypoderm, composed chiefly of plasma cells and lymphocytes, with a few productive elements, epithelioid and giant cells, the latter noticeably less than in tuberculosis verrucosa. Between the cells and running generally perpendicular to the surface is a network of fine fibers, the lupus reticulum, which is composed of elastic fibers, chiefly because of their greater resistance. The giant cells contain the bacilli which average about one in 200 sections examined. Epithelium remains intact, slightly acanthotic. This is the simplest form of the disease. As in tuberculosis of the lungs, adventitious elements change its complexion somewhat. There is a catarrhal lupus in which coccic infection causes a masking of the lupoma by leucocytes with a thinning or loss of epithelium, crust formation and ulceration (lupus exulcerans). In fibroid lupus, the toxin causes proliferation of fibroblasts with formation of new connective tissue. Giant cells are more numerous and other elements fewer in this type. Acanthosis is marked and forms the basis of the malignant epithelioma which sometimes complicates the disease. Lupus verrucosus shows a hyperkeratosis over the lupoma. Necrosis is no part of the process *per se.* Direct inoculation is rare in causation.

Scrofuloderma and *Scrofulous Gumma* are tuberculoses of the hypoderm, infection being carried to it by lymphatics from subjacent nodes or bone. Tubercle bacilli are not easily demonstrated except by inoculation into animals. The disease is almost a pure exudation of lymphocytes and plasma cells about lymphatic vessels; productive elements are excessively rare. Infiltrated areas undergo early a liquefactive necrosis and small, sterile cold abscesses are formed. If unchecked, the disease extends to the surface and breaks through, leaving indolent ulceration with fistulous tracts to the original focus. Thrombosis is not a feature of this type of tuberculosis.

Tuberculosis Vera (orificialis, miliaris) occurs about the mucous outlets from inoculation of bacilli in visceral discharges. It is superficial, the only cutaneous disease showing formation of true tubercle, ulcerates readily and contains myriads of organisms.

Lichen Scrofulosorum is a perifollicular disease, occurring always in the tuberculous, in which it is almost impossible to demonstrate bacilli. Inoculation fails in a great majority of instances. Either the toxins alone are at work here or the process once started continues after the bacilli have disappeared.

Color is lent to the former theory by the fact that injections of tuberculin have caused outbreaks. Histologically, lichen scofulosorum is a productive tuberculosis with little tendency to necrosis.

Erythema Induratum Scrofulosorum is unquestionably a tuberculosis of the hypoderm, the same remarks as to finding bacilli being applicable here as in the lichen of the scrofulous. Inoculation has been successful in only two or three cases. While examination shows an exudative condition generally, productive elements may at times occur in large proportion. Lymphatics are chiefly involved and cold abscesses are formed as in scrofuloderma from which erythema induratum is differentiated by a pronounced thrombotic tendency in the smaller vessels. The infiltration extends to the skin and causes ulceration at times.

Leprosy is seen in three forms, the nodular, anesthetic and mixed. All are produced by Hansen's bacillus which resembles that of tuberculosis in almost every particular and which gains entrance, according to his and Morrow's observations, by the nasal passages. The *nodules* of leprosy resemble a productive tuberculosis; lymphocytes and plasma cells are few, the bulk of the lesion being made up of vacuolated and multinucleated cells (lepra cells). Bacilli are found in them and free in the tissues in great numbers. In *anesthetic* patches, bacilli are much fewer, infiltration is slight and the chief changes are found in the sensory nerves, especially the cutaneous filaments showing marked degeneration. The bacilli are easily seen in the peri- and endoneurium. Their first effect is to cause a parenchymatous neuritis which in its later stages becomes also interstitial and involves motor nerves as well. Mixed leprosy combines both forms. There are no changes in the central nervous system.

Rhinoscleroma is a granuloma affecting the nasal and throat mucous membranes and adjacent skin. It is caused by an encapsulated organism which is easily found and conforms entirely to the characters of Friedländer's pneumococcus. The disease shows the usual features of granuloma, lymphocytes, plasma, epithelioid and a very few giant cells without regular arrangement and traversed by threads of fine connective tissues forming a reticulum. Necrosis rarely occurs.

Blastomycosis is primarily a skin affection, from whose foci systemic infection may take place. Its organism is a yeast fungus, the blastomyces described by Busse and Gilchrist independently, a double-contoured round body about 10 μ in diameter, which stains readily with methylene blue and can be cultivated on almost any medium. Outside the body, it forms a mycelium, inside it increases by budding as do the yeast fungi generally. The cutaneous lesion is singularly like that of tuberculosis verrucosa, clinically and pathologically, except that it ulcerates rather readily. The cutis and hypoderm are invaded by the same productive granulomatous process, there is an overgrowth of the epithelium, both acanthosis and hyperkeratosis with abscesses at the tips of the elongated rete pegs. Blastomycetes are found free, in the abscesses and in the substance of giant cells.

Granuloma Fungoides (*Mycosis Fungoides* of Alibert).—In spite of its multiform clinical characters, granuloma fungoides shows the same pathological picture from beginning to end. It has certain remote relationship to sarcoma but it never forms metastases in nodes or viscera. The finding by McVail of a white bacillus has not been confirmed. Bacteria have been recovered often but are regarded as contaminations.

The process confines itself strictly to the upper part of the corium. There is congestion and exudation of small numbers of white corpuscles, mononuclear predominating, but the mass of the cells is made up of bizarre forms derived from fibroblasts. Some are small, polyhedral from pressure with acidophile protoplasm and a vesicular nucleus, others are larger and distinctly epithelioid and giant cells partly or fully formed appear scattered irregularly, the whole traversed by a fibrous reticulum. The giant forms are not numerous. The epithelium shows catarrhal changes with scale formation, rarely crusts. When the cell mass becomes large, its central and superficial portion dies, possibly as a result of pressure on the vessels. Necrosis is followed by absorption or exfoliation of epidermis and ulceration. Large tumors occasionally undergo complete absorption, leaving the corium weakened in resisting power. The epithelium at times assumes a proliferative action which microscopically cannot be distinguished from squamous-celled epithelioma. Granuloma fungoides invariably results in death from cachexia, if not from intercurrent disease.

Xanthoma is a name applied to several yellow tumors only one of which properly belongs here. *Xanthoma palpebrarum* is caused by a fatty degeneration of the muscles of the eyelids, its only situation. *Xanthoma tuberosum* or *vulgare* is a near relative of lipoma according to Török, a development of fat in a heterotopic situation, the corium. The cells in which the fat droplets are contained are large mono- or multinucleated cells ("xanthoma cells") undoubtedly of connective tissue origin. Inflammation is not present except under irritation. Tuberose xanthoma is sometimes accompanied by jaundice which is thought to be obstructive since the tumors have been found in the bile-ducts. *Xanthoma diabeticorum* is a granuloma possibly due to a toxemia occurring in diabetes and in its allied state, obesity. It first appears as a subacute inflammation about the vessels of the reticular, the exudation composed of lymphocytes and fewer plasma cells. An added element soon appears in the shape of mast and epithelioid cells. The endothelium of the capillaries is swollen and in its cells, or in the epithelioid, fatty degeneration occurs. The fat breaks through the cell wall and escapes into the surrounding tissue where it lies in tiny droplets or collects in larger masses. In alcohol specimens where the fat is dissolved, empty spaces are seen traversed by protoplasmic filaments, remains of degenerated cells. The only connection between the three forms is the presence of fat in the cutis which gives the characteristic yellow color.

Lupus Erythematosus.—The disease is an inflammation with an ultimate necrosis and death. Its relationship to the granulomas is problematical; its connection with tuberculosis cutis even more so. Great efforts have been made in latter years to prove its tuberculous origin, a theory which is particularly strong in France. Many cases do occur (more than a majority) among the tuberculous. No organism has ever been recovered from the tissues.

Erythema, which means congestion and exudation, is the first change, always about the superficial vessels. There is a fair amount of edema followed by the usual epidermic phenomena. Acanthosis results, with scale formation and keratotic prolongations into the openings of the pilo-sebaceous follicles. Except in the earliest stage when it appears about the vessels, there is no regular arrangement of the cellular infiltration. It is confined to the papillary body and upper part of the reticular layer and consists of lymphocytes, plasma cells, very few leucocytes and fibroblastic derivatives chiefly of the epithelioid type. Capillary thrombosis occurs simultaneously with the attainment of the height of the infiltration. The walls of the vessels appear to be intact but the clot blocks them completely. Lack of nutrition causes a degeneration not unlike coagulation necrosis in which not only the infiltration but the skin appendages disappear. The thinned epidermis is left covering an atrophic depressed area from which the detritus has been absorbed. Capillary obstruction brings about a permanent dilatation of the vessel near the thrombosed area.

Parasitic Diseases.

Animal Parasites.—Description of these parasites is properly confined to works on zoölogy. Their number includes those which live on the skin like the *pediculus capitis, corporis* and *pubis*, varieties of the same species and those which pursue at least part of their lives in its substance like the *acarus of scabies*, the female burrowing in the epidermis and leaving in the furrow a trail of ova and feces, or the *demodex folliculorum*, a segmented worm which has its habitat in the sebaceous glands and sometimes causes a brown pigmentation. All are capable of causing irritative inflammation.

Vegetable Parasites include the ringworm fungi, the achorion of favus and the fungi of pityriasis versicolor and erythrasma.

Ringworm Fungi.—There are several varieties of the fungus, chief of which are the large- and small-spored. Both are concerned in the production of ringworm of the scalp and body, as a rule, the large-spored alone in ringworm of the beard. Differentiation lies not so much in the size of the spores as in their arrangement. The small species sheaths the hairs, is rarely seen in mycelial threads and being closely packed appears in the form of a mosaic. The large-spored fungus occurs outside and inside the hair (ectothrix and endothrix) in chains, "rosary" arrangement. They can be demonstrated as can all fungi by placing the hairs or scales in a dilute solution of caustic potash. They may be stained in a solution of gentian violet in aniline water, decolorizing by Gram's method. Both may cause intense exudative inflammation. Cultural characters differ in some particulars. (Pt. I., pp. 202–204.)

CHAPTER X.

DEATHS BY VIOLENCE AND POISON.

Lesions in Asphyxia.

THE blood is dark and usually fluid, the hypostatic congestion is dark, face cyanotic, organs in condition of passive hyperemia. Respiratory mucous membrane in parts or generally injected and bronchi contain frothy, blood-stained mucus, especially if the fatal process was slow. The lungs contain more dark blood if process rapid, with subpleural and subpericardial bleeding; partial emphysema is common. Right side of heart and great veins distended with blood, left side may be empty, but if heart action lasted longer than respiration this may not be so. Hanging and choking cause pressure on the carotids, jugulars and vagi; the vessels may be firmly occluded. If the body was immediately cut down there may be no line on the neck made by the cord or rope, otherwise it may be well marked. After hanging the line is evident on the sides of the neck and is lost near the ears; if something was tied round the neck its course is usually horizontal. The skin may show abrasions which turn dry and semi-transparent after death; extravasations may be microscopic. When the death followed choking by the hand, impressions of the fingers and nails, ecchymosis under the skin or in the tissues, fracture of the larynx—rarely of the hyoid—may be found. In both hanging and strangling partial erection of the penis, escape of semen (into the urethra), urine or feces may occur. Drowning produces the same general conditions, the nails and hands may contain sand, mud or weeds; similar matter may be found in the stomach; the fluid may have entered the respiratory passages ; microscopical examination of such fluid and of the water in which the death occurred may give valuable evidence in medico-legal cases. The lungs may be distended so as to cover the heart and overlap. In recent cases the extensor surfaces of the limbs may show marked cutis anserina, and the scrotum may be shrunken. In recently born infants the presence of a dried umbilical cord, still attached, may be of importance ; the back of the tongue and the opening of the larynx may show finger-nail wounds. Foreign bodies in the air passages are usually firm material, portions of food, bits of cloth, etc., introduced by accident or design; the latter often in infants. Vomited matters from the esophagus may also close the larynx or trachea, as during coma from alcoholism. In the new-born cessation of placental circulation during parturition may excite premature efforts at respiration ; the subserous hemorrhages in heart and lung are commonly present, and, as in the above cases, froth and foreign material occur in the bronchi.

Other appearances occasionally seen in death from asphyxia are protrusion of the tongue, injected conjunctivæ, ecchymoses on the face, neck and chest.

The distinction should be made between *suffocation*, prevention of air entering the lungs by pressure on the air passages or cloths and other matters held over the nose and mouth, *strangulation*, which means constriction of the neck by cord-like materials, and *asphyxia*, which is the condition of the respiration produced thus and also by irrespirable gases. It is always possible that a death by other violence or poison may be concealed by hanging the body or throwing it into the water ; interpretation of post-mortem conditions should therefore be cautious.

Burning causes death by involving a large extent of surface or by secondary conditions following this. The inhalation of smoke, hot air and flame, incautious ingestion of hot fluids, and inhaling steam may also be fatal. The greater part of the surface may be destroyed, and if by flame the superficial hairs are crisped, swollen at the ends, or lost ; in scalds they may be preserved for a time. The greater part of a limb may be completely charred, or the whole body. The skin is dry, red and hard in less severe cases, or suppurating ; in the mildest forms redness and vesicles are formed. The blood cells may be destroyed, with hemoglobinemia.

Congestion and edema of the respiratory passages, often croupous, and of the cranial and abdominal viscera, serous inflammations (pleura and peritoneum) and perforating ulcer of the duodenum with general catarrhal gastro-enteritis, may all be found if the patient survives the first shock of injury.

Electricity, either from currents or lightning, may cause superficial burns, and with the latter there are branched markings on the skin, edema and laceration, injury to important viscera, and occasional fractures of bones. There are no characteristic and constant lesions in these cases.

Other deaths by violence show the wound or injury to important viscera (brain), fractures, etc.

Poisons.

Alkalies.—The commonest alkalies causing death by accident or suicide are ammonia, potash and soda. Parts which have been in contact with the chemical are usually swollen and softened, congested and inflamed ; black from changes in the blood and peeled off in later stages. These effects are commonly found about the mouth, pharynx, esophagus and stomach. With a stomach full of food the poisonous fluid may ride over to the pylorus and enter the duodenum, while most of the stomach escapes. Inhalation of ammonia causes intense congestion of the respiratory mucous membrane. Death may follow in a few hours or weeks ; recovery may occur with contracting scars in parts attacked.

Acids : Mineral. *Hydrochloric.*—Whitish eschars on parts in contact with acid, false membranes may form ; later sloughing and inflammation. Death may occur in a few hours ; if later than twenty-four hours, fatty degeneration of kidneys and suppurative inflammation along track of acid may develop. Blood may be fluid or thickened.

Nitric.—Yellow eschars (xanthoprotein) in mouth, esophagus and stomach, intense hyperemia and inflammatory reaction, stomach contains a mixture of blood and necrotic tissue, yellow or greenish; it may be perforated.

Sulphuric.—Eschars gray or black, mucosa of esophagus black and peels off in shreds, stomach contains black, sticky material, its walls congested and hemorrhagic, rugæ marked and partly destroyed, perforation may occur.

All these poisons depend for their effect upon their concentration and the time during which they act. By accidental entrance to the air passages they produce similar destruction. Their effects often skip a part of the tract from mouth to intestine, being manifest at times only in the lower esophagus, stomach and duodenum. They may retard putrefaction in the rest of the body.

Acids: Organic.—*Acetic, oxalic* and *tartaric acids* may cause death, the second most commonly. They may be rapidly fatal or excite inflammation, ending fatally or with scar formation and recovery. The mucous membranes attacked may be hyperemic and inflamed, with oxalic acid commonly white and shrunken, easily stripped off. In the stomach disorganized blood, mucus and necrotic tissue, swelling, opacity and softening of the mucosa, inflammation and gangrene may occur.

Carbolic.—The parts attacked are whitened and condensed, there are hemorrhages along the track of the poison, the rugæ of the stomach and the intestinal folds (*valvulæ conniventes*) are thickened and have a peculiar worm-like feel through the unopened organs. The color of the serous surface opposite the action of the acid varies from pink to brick-red, and neighboring organs may show patches of the same tint. The urine may be dark green and its odor, like that of the stomach, is characteristic of the acid. Other organs are usually congested. Death may occur instantly from nervous shock, or later from destructive chemical effects. If the stomach contained food (especially with alcoholic beverages) it may escape the action.

Hydrocyanic.—Death may be immediate and only venous congestion, fluid blood, pinkness in the stomach and gut, and the odor of the drug (fruit-like) give evidence of the nature of the case. With potassium cyanide corrosive action occurs, as with alkalies.

Metallic Poisons. *Antimony, Usually as Tartar Emetic.*—In acute cases gastro-enteritis occurs, sometimes peritonitis. In chronic cases there may be no marked lesions.

Arsenic.—Inflammation of the stomach, the presence of green particles (Paris green) about the mouth and in the stomach, or white grains of arsenous acid, extravasation of blood, enteritis, swelling of the lymphoid tissues in the canal, peritonitis, nephritis and cystitis may occur. Fatty degenerations in viscera are common. The body may be preserved by the poison.

Copper.—Inflammation of the gastro-intestinal tract, ulceration and necrosis of its mucosa, perforation and peritonitis are the usual results. A green color may be noticed in the parts.

Lead.—The metallic salt in substance (often acetate) may be found in the inflamed stomach and intestines. In chronic cases degenerations of muscular and nervous tissue and chronic inflammation of meninges may be added to intestinal lesions. Nephritis and gout may be associated with these.

Mercury.—The bichloride is the common salt used. The mucosa of parts affected is swollen, whitened, hyperemic; later ulceration may occur. Nephritis is usual if life is prolonged. Severe necrotic inflammation of the colon (diphtheritic colitis) is observed in chronic cases.

Phosphorus.—Acute cases present hemorrhage and inflammation in the stomach, the poison in substance (match heads) and a peculiar odor. Vomit, contents of alimentary canal, and feces may glow in the dark. After a few days icterus, subserous and submucous hemorrhages, fatty degeneration of the liver and other viscera, with bile staining, are commonly developed. The liver is particularly affected and may resemble the liver of acute yellow atrophy.

Carbonic Oxide.—The characteristic effect of this poison is the cherry-red color of the blood, hypostatic congestion of the surface being also of this color. In some cases there is cerebral hemorrhage from a small branch of the middle cerebral artery. The spectrum of the blood and the corroborative tests should be used to determine the presence of CO Hb. (See p. 53.)

Sulphuretted Hydrogen.—Poisoning occurs from working in sewers and privy vaults where the gas collects, and in rare cases from large production in the intestines; the gas may be discovered in the urine. The viscera smell of the gas, the blood is fluid and black, like ink, and vascular organs (lungs) may also be dark colored. This color seen through the skin gives the surface a green or blue-green tint. Rigor mortis may be absent, the viscera are usually hyperemic.

Alkaloids of various kinds may cause death, but their presence requires careful chemical analysis of stomach and intestinal contents. If the crude form of the drug is used microscopic examination of the contents of the stomach and intestines, and of the feces, may reveal leaves, roots and seeds, or their fragments; these may be sufficiently characteristic to identify the poison.

APPENDIX TO CHAP. X. ALPHABETICAL SYNOPSIS OF LESIONS AFTER CERTAIN INFECTIONS AND EXANTHEMATA.

Anthrax.—Malignant pustule on the surface or diffuse anthrax edema, rigor mortis marked, blood dark cherry red, not much coagulated, diffuse or circumscribed hemorrhages in cerebral membranes, at times small foci of bleeding in brain, catarrhal inflammation of alimentary tract, blood-stained fluid in body cavities and hemorrhages in serous membranes, spleen moderately or much swollen, soft and of very dark red. If infection took place through the alimentary mucosa, edema, hemorrhage in tissues and pustules may occur in pharynx or anywhere along the tract. Bacilli found about port of entry, in edematous connective tissue, and in blood capillaries.

Bubonic Plague.—Petechial and larger ecchymoses in skin, mucous and serous membranes, boils and erysipelatous dermatitis, primary carbuncle at point of infection, secondary ones elsewhere. Hyperemia of spleen and kidneys, pneumonia and red infarcts of lungs. Chief lesions in lymph vessels: superficial nodes swollen with edema and bleeding in surrounding tissues; later, suppuration and external perforation of the nodes; commonest affected, inguinal, crural, axillary, cervical, the deeper ones may be a little affected. Bacillus in blood, abscesses and organs.

Malaria.—Spleen very large, hyperemia and hyperplasia at first, induration and pigmentation in chronic forms, rupture may occur in former condition with hemorrhage into peritoneum; liver swollen and hyperemic, pigment granules in and about capillaries; in some cases miliary collections of leucocytes, epithelial cells and pigment with fibrosis; kidney pigmented, epithelia degenerated, capillaries may contain plasmodium. Melanemic pigment also in bone marrow and brain.

Morbilli.—The skin lesions may have disappeared. Exudative inflammations of respiratory tract, bronchitis and pneumonia, commonest complication and cause of death. Hemorrhages in various surfaces in very severe forms. Cloudy swelling of viscera.

Pyemia.—Results of distribution of pyogenic organisms through the blood, with suppuration in joints and serous cavities, abscesses in viscera and muscles; but these lesions may be limited to a few organs, as the spleen and kidneys. Various degenerations of the visceral epithelium and lesions of complicating diseases, typhoid and scarlet fever, cholera and puerperal infection. The original focus of pus may be discovered, as otitis media, phlebitis in a superficial vein, etc. Post-mortem changes occur rapidly, icterus is common, the blood is fluid and brownish-red, rigor mortis may not be well developed.

Rabies.—Rigor mortis marked, face may be cyanotic, the wound which was the point of infection may not be found, or a scar firm and apparently normal. In recent cases the lymph nodes near the wound may be edematous and hyperemic. The meninges of cord and brain may be very hyperemic, as also the brain tissue; small hemorrhages may occur in the central nervous organs and extravasations about the origin of the pneumogastrics have been found. The pharyngeal mucosa is congested and the tonsils swollen; alimentary mucosa may be swollen with punctate hemorrhages. The same may occur in the respiratory passages with inspiration pneumonia from dysphagia. Liver and spleen hyperemic with parenchymatous degeneration.

Relapsing Fever.—Spleen strongly swollen and hyperemic, may be ruptured, dark colored and soft; may be red infarcts. All other organs and voluntary muscles show acute parenchymatous degeneration; fatty changes in the heart may be marked. Intestinal canal shows hyperplasia of lymphoid tissues. Organism in blood and spleen for hours after death.

Scarlatina.—Skin pale, desquamating in scales or in large epidermal leaves, may form casts of fingers. Otitis media, acute nephritis, hemorrhages in skin and membranes, meningitis and other complicating lesions may be present, with edema of face and legs.

Septicemia.—Putrefaction rapid, blood fluid and thin, hemorrhages on serous and mucous surfaces, cloudy swelling of organs and fatty degeneration. About the source of infection thrombosis and gangrene may occur or a purulent focus may be found. Various pyogenic organisms may be recovered from the heart's blood, exudate and organs, but many cases give negative results.

Typhus Fever.—Emaciation, gray color of surface, eruption not found, small hemorrhages in skin as small bluish-red spots over the whole body or chiefly the lower extremities ; these may be merely pigmented spots. Rigor mortis marked but disappears early, putrefaction rapid, muscles brown and dry with waxy degeneration at times, hyperemia of meninges ; spleen swollen, red and soft, fatty degeneration of heart and renal epithelium and liver cells ; gall-bladder contains little thick and dark bile. Inflammatory changes in respiratory and alimentary mucosa, bronchitis, pneumonia, gastro-enteritis, etc.

Variola.—The eruption persists after death and may be found in any stage, other lesions are those of high fever and complications.

Yellow Fever.—Rigor mortis well marked and appears early, muscles dark and dry, skin yellow or very dark yellow green, subserous hemorrhages in lungs and heart, blood may be thin and fluid. Gastric mucosa strongly congested and swollen, hemorrhages into the membrane and in the cavity are common ; the blood dark and thick (coffee grounds) ; in the upper part of the small intestine similar lesions may be found, in the colon dysenteric necrosis may occur. Liver swollen, or moderately decreased, soft, color varies from dark red to light yellow with bile staining ; gall-bladder contains a small amount of dark thick bile. Spleen may be small. Kidneys show acute nephritis, often hemorrhagic.

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